ARCHIVES OF OTOLARYNGOLOGY

VOLUME 38 JULY 1943 NUMBER 1

COPYRIGHT, 1943, BY THE AMERICAN MEDICAL ASSOCIATION

SARCOMA OF THE TONSIL

IMPRESSIONS MADE BY SEVEN CASES

C. A. WHITCOMB, M.D., M.Sc. (MED.)
PHILADELPHIA

Sarcoma of the tonsil is not a common tumor. The incidence is well illustrated by reports from the Memorial Hospital [1] and the Mayo Clinic [2] which show, respectively, 22 and 55 cases of lymphosarcoma during a period of five years. No case of reticulum cell sarcoma is mentioned in these reports.

The family physician, whom the patient consults first, will encounter, therefore, few cases during his professional life, and even the otolaryngologist attending a general hospital clinic will probably not see this tumor frequently enough to become familiar by personal experience with the appearance and behavior of sarcoma of the tonsil.

Sarcoma of the tonsil should not be confused with carcinoma of the anterior faucial pillar. The commonest type of malignant growth of the region of the tonsil is squamous cell carcinoma, grade 2 or 3, involving the anterior pillar and any or all of the following adjacent structures: the gum, the cheek, the soft palate, the tonsil, the floor of the mouth and the margin of the tongue (fig. 1). The normal tonsil in old men, who are the victims of this disease, is atrophic and more or less hidden by the anterior pillar. On the side of the disease, it is usually difficult or inexpedient to expose the tonsil itself sufficiently to determine the extent to which it is involved by the carcinoma of the pillar. It is usually presumed, however, that the tonsil is involved.

This tumor should be classified, in my opinion, not as a primary neoplasm of the tonsil but as a carcinoma of the pillar. The difference in the course of carcinoma of the pillar and that of sarcoma of the tonsil justifies the drawing of this distinction. Carcinoma of the pillar is a slowly growing tumor, which almost never metastasizes beyond the regional lymph nodes of the neck. It kills by pain and starvation. Sarcoma of the tonsil grows rapidly, spreads quickly and widely and kills by visceral metastasis. Of greater importance than this difference in method of growth is the fact that small cancers of the anterior pillar can be cured by surgical excision (fig. 2). The failure to report separately these two types of malignant tumors obscures the results of treatment obtained with squamous cell carcinoma of the pillar and with sarcoma of the tonsil.

The cause of sarcoma of the tonsil is not known. Tobacco, chronic irritation and syphilis, which are usually given as contributory causes of cancer of the mouth, are not suspected of playing any part in the origin of sarcoma of the tonsil. It is not preceded by leukoplakia.

From the Jeanes Hospital, Philadelphia.

Read at the joint meeting of the Section on Otolaryngology of the College of Physicians, and the Philadelphia Laryngological Society, Oct. 22, 1942.

1. Martin, H. E., and Sugarbaker, E. L.: Cancer of the Tonsil, Am. J. Surg. **52**:155-196, 1941.

(Footnotes continued on next page)

Mallory [3] expressed the belief that all types of lymphoblastoma (lymphocytoma, lymphoma, lymphosarcoma, pseudoleukemia, lymphatic leukemia, Hodgkin's disease, plasmocytoma and giant lymph follicle hyperplasia) are different manifestations of one disease. The rapidity of the appearance of widely scattered,

Fig. 1.—Squamous cell carcinoma of the anterior faucial pillar. This tumor, representing the commonest type and location of malignant growths in the region of the tonsil, is to be distinguished from malignant tumors primary in the tonsil. *A* indicates the uvula; *B*, the soft palate; *C*, the anterior pillar; *D*, the cheek, and *E*, the posterior wall of the oropharynx.

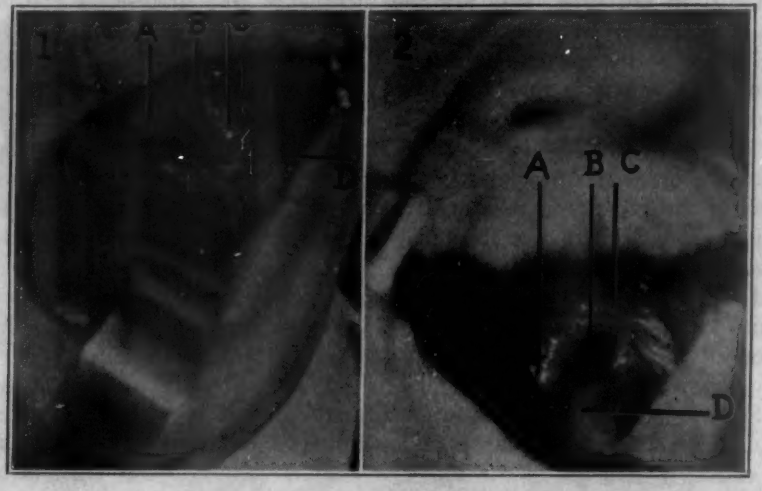


Fig. 2.—Squamous cell carcinoma of the anterior pillar. *1*, early stage, grade 1; treatment by excision of the pillar. *A* indicates the region of the uvula; *B*, the soft palate; *C*, the anterior pillar, and *D*, the cheek. *2*, five years after operation. The patient has not had a recurrence for ten years. *A*, is the uvula; *B*, the posterior wall of the pharynx; *C*, the soft palate, and *D*, the margin of the tongue.

2. New, G. B.; Broders, A. C., and Childrey, J. H.: Highly Malignant Tumors of the Pharynx and Base of the Tongue, Surg., Gynec. & Obst. **54**:164-174, 1932.

3. Mallory, F. B.: Principles of Pathologic Histology, Philadelphia, W. B. Saunders Company, 1914, p. 326.

simultaneous metastases of sarcoma of the tonsil and the distribution of tumor masses sometimes observed at autopsy causes one to recall Mallory's opinion and to think of a generalized disease of the lymphoid tissue or of a tumor with multiple sites of origin rather than metastases from a single site of origin.

Knowledge of the topographic anatomy of the tonsil is essential to an understanding of the signs and symptoms of sarcoma of the tonsil and is useful in the administration of treatment. The top of the tonsillar fossa is formed by the junction of the faucial pillars and the soft palate; the fossa is continuous below

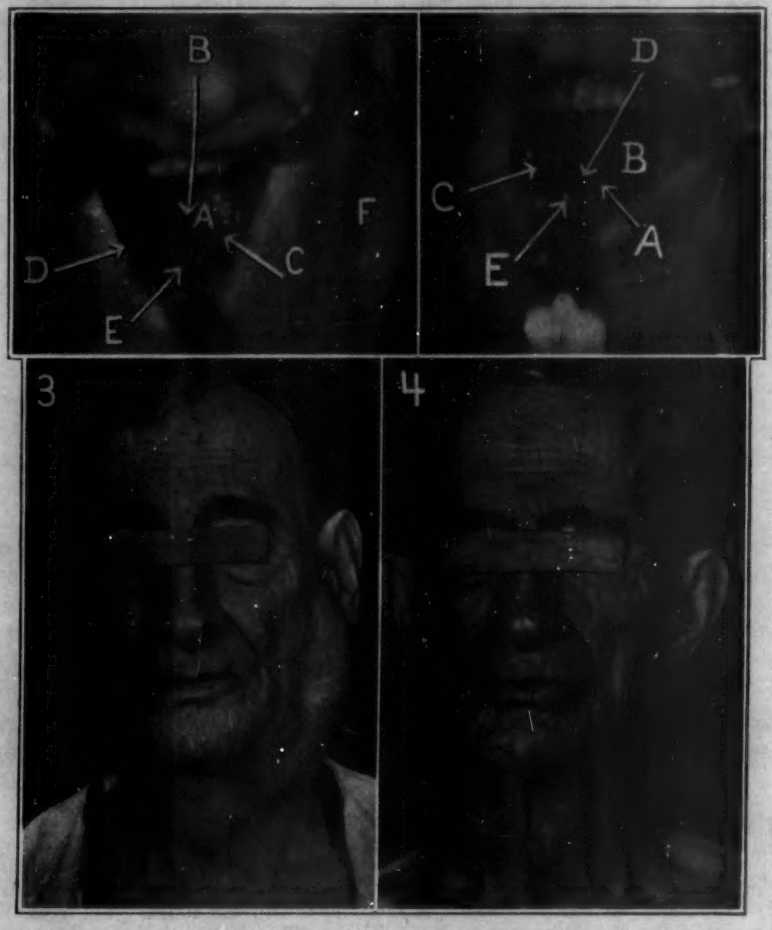


Fig. 3.—Reticulum cell sarcoma of the left tonsil. *1*, before treatment. *A* indicates the upper part of a large sarcoma distending the tonsillar fossa, displacing the soft palate (*B*), the anterior pillar (*C*) and the uvula (*D*). *E* indicates the site of removal of a biopsy specimen; and *F*, an unusually large metastasis at the angle of the jaw. *2*, three weeks after implantation of radon seeds in the sarcoma of the tonsil. *A* indicates the tonsillar fossa; *B*, the anterior pillar; *C*, the uvula; *D*, the posterior pillar, and E, the tongue. *3*, before treatment, with an unusually large mass at the left angle of the jaw. *4*, three weeks after the beginning of roentgen ray treatment of the mass in the neck.

with the vallecula. The tonsil is clamped directly to the middle constrictor muscle of the pharynx, which separates it from the carotid sheath. It has no direct attachment to the anterior pillar or to the plica triangularis. The limitations of the upper pole of the tonsil are more distinct than those of the lower pole, where,

if lymphoid tissue is abundant, no line of division between the faucial and the lingual tonsil may be discernible.

A sarcoma of moderate size involving the upper pole distends the supratonsillar fossa, and a large sarcoma in this location causes bulging of the soft palate and displaces the uvula, thereby producing an appearance suggestive of invasion of these structures (fig. 3*I*). It also compresses the tissues about the orifice of the eustachian tube. Enlargement of the lower pole by tumor makes contact with the tip of the uvula and the margin of the epiglottis. A large tumor of the lower pole of the tonsil also causes elevation of the tissues below the angle of the jaw (fig. 4), which may be mistaken for a fixed metastasis in a lymph node.

Fig. 4.—X-ray apparatus used for intraoral treatment. Roentgen rays pass through the lead cone (*A*), the hidden end of which is in contact with the tonsil. The cone is used to protect the rest of the mouth from roentgen radiation. The diagnosis is transitional cell carcinoma of the left tonsil. The mark on the skin indicates the size and position of the large swelling at the angle of the jaw commonly present with any histologic type of malignant tumor of the tonsil. The swelling is produced partially or entirely by tumor of the lower pole of the tonsil.

Only the middle third of the tonsil can be seen by depression of the tongue. A laryngeal mirror must be used to see the lower pole of the tonsil and a pillar retractor to expose the upper pole. The surface marking of the lower half of the tonsil is an area on the skin between the angle of the jaw and the great wing of the hyoid bone.

The tonsil consists of three types of tissue: squamous epithelium, reticular connective tissue and lymphocytes. The cell bodies and delicate fibrils of the

reticular connective tissue of the tonsil cannot be seen until the overlying lymphocytes are dislodged. Neoplasms of the tonsil are composed of cells tending to differentiate into one of the three types of normal cells. They are classified as squamous cell carcinoma, transitional cell carcinoma, lymphoepithelioma, reticulum cell sarcoma and lymphosarcoma.

Sarcoma of the tonsil is composed of cells tending to differentiate into lymphocytes or into the cells of the supporting reticulum of the lymphoid tissue. In lymphosarcoma the loss of normal architecture of the tonsil (absence of germinal centers as seen by low magnification) is more easily recognized than the departure of the tumor cells from the structure of the normal lymphocyte. Differential staining of the fibrils aids in the diagnosis of reticulum cell sarcoma.

Carcinoma, which can arise only from the epithelium covering the exposed surface and crypts of the tonsil, is often so anaplastic that pathologists may not agree on the diagnosis. New and associates [2] stated that squamous cell carcinoma grade 4 is often called lymphosarcoma or Hodgkin's disease. The degree of differentiation of the most mature cells of any neoplasm determines the ease and certainty of recognition of the type of neoplasm. Many neoplasms of the tonsil are composed of cells so lacking in signs of maturity that differential diagnosis may be difficult. Stewart,[1] pathologist to Memorial Hospital, New York, in referring to the differential diagnosis of lymphosarcoma and lymphoepithelioma, stated: "the field of diagnosis of these tonsillar and nasal pharyngeal tumors is one of the most difficult in tumor pathology."

The word "sarcoma," which is derived from a Greek word meaning "flesh," indicates the outstanding feature of this type of neoplasm. Sarcoma of the tonsil has the same gross characteristics as sarcoma arising in other parts of the body. It appears as a bulky, rapidly enlarging, elastic mass, which displaces adjacent tissue.

When a patient with sarcoma of the tonsil first consults a physician, examination shows a normal throat, except for a large, rather normal-looking tonsil. The faucial pillars and the soft palate are normal unless congested by the pressure of a massive tumor. If spontaneous ulceration occurs, it does so infrequently. On the patient's admission to the hospital, an ulcer, due to misguided therapeutic incision or to the excision of a biopsy specimen, is often present.

Metastases from sarcoma of the tonsil play a more vital part in the welfare of the patient than does the primary neoplasm. Ewing,[4] in a general discussion of sarcoma arising from all types of connective tissue, stated:

> . . . it is characteristic of the disease that, with rare exception, the embolic cells travel through the blood vessels. Except in lymphosarcoma, the occurrence of metastases in lymph nodes justifies the suspicion that the tumor is not a sarcoma.

Six of the 7 patients whom I have seen appeared with a mass just below and behind the angle of the mandible (table). The typical mass (fig. 4) is large, deep and immovable and is probably the tumor of the lower pole of the tonsil, but may consist of the tumor of the tonsil and superimposed lymphadenopathy. This type of mass, which can be seen across the room, is so constant as to form the most characteristic clinical feature of the disease.

The blood vessels may well furnish the pathway by which sarcoma of the tonsil reaches the chest and abdomen. One usually thinks of malignant tumors of the tissues of the head and neck as metastasizing to the lungs before spreading to other parts of the body. Sarcoma of the tonsil appears to be an exception in this respect. Metastases to the abdomen occurred without metastases to the chest in 4 of 5 cases in which a roentgenogram was taken three to eight weeks before death;

4. Ewing, J.: Neoplastic Diseases, Philadelphia, W. B. Saunders Company, 1940, p. 270.

the roentgenogram of the chest in the fifth case showed metastatic masses. One can confidently expect to find on physical examination abdominal masses in the late stage of this disease.

The signs of sarcoma in the tonsil are more prominent than the symptoms. As a rule, to which there are some exceptions, sarcoma confined to the tonsil and cervical lymph nodes is not a painful disease. The absence of pain leads to procrastination and prolongs the period of office treatments by the family physician. Most of the early symptoms can be explained solely by enlargement of the tonsil. A few patients discover a mass in the throat; others complain of symptoms of a foreign body due to enlargement of the lower pole of the tonsil, but most patients note an essentially symptomless lump in the neck. Pain is present in the late stage, which is characterized by thoracic, abdominal or skeletal metastasis.

Trismus is not a symptom of sarcoma of the tonsil, but occurs as a complication of squamous cell carcinoma of the faucial pillar which has penetrated the

Data on Seven Sarcomas of the Tonsil

		Description of Disease					Outcome of Disease		
							Time of Death		
Age, Yr.	Sex	Microscopic Diagnosis	Complaint of Patient	Duration of Complaint, Mo.	Mass in Neck, Cm.	Previous Treatment Advised or Given	After Treatment, Mo.	After Onset, Mo.	Cause of Death
48	F	Reticulum cell sarcoma	Mass in neck Mass in throat	14 6	5	Tonsillectomy	36	50	Abdominal metastasis
78	F	Reticulum cell sarcoma	Mass in throat	3 wk.	No mass	"Electric needle"	22	24	"Hematuria"
74	M	Reticulum cell sarcoma	Mass in neck	6	6	Ointments	2½	8	Weakness and abdominal pain
71	M	Reticulum cell sarcoma	Sore throat	3	8	Gargles	6	9	Hemorrhage from throat
66	M	Reticulum cell sarcoma	Mass in neck	1½	5	Gargles	10	12	Intestinal obstruction from abdominal metastasis
57	M	Reticulum cell sarcoma	Sore throat	2	2	Incision for "quinsy"	1	4	Myelitis; metastasis to vertebrae
62	M	Lymphosarcoma	"Throat trouble"	6	5	Tonsillectomy	18	23	Metastases in lungs and abdomen

wall of the pharynx and has involved the internal pterygoid muscle by inflammation or cancer.

Pain deep in the ear, which probably arises from irritation of the sensory end organs of the glossopharyngeal nerve and reaches the tympanic plexus by Jacobson's nerve, is a common symptom of squamous cell carcinoma of the base of the tongue. Pain in this location is not present in cases of sarcoma of the tonsil.

DIAGNOSIS

Sarcoma of the tonsil should be differentiated from various lymphoblastomas of the tonsil by examination of the blood and the rest of the body and from other types of malignant tumor of the tonsil by microscopic examination of a suitable biopsy specimen.

The family physician does not quickly recognize sarcoma of the tonsil. He mistakes it for "trench mouth," "Vincent's angina," "quinsy" or "gumma." Tonsillectomy is sometimes done for "infected tonsils" and sarcoma is reported by the pathologist, to the surprise and chagrin of the unsuspecting clinician. A mass in the neck unaccompanied by symptoms of trouble in the throat is called

"an abscess," "a cyst" or "a benign tumor" and is incised, excised or poulticed. Teeth are usually extracted.

Some of these errors in diagnosis can be understood. A particular sarcoma at first glance may suggest quinsy; all necrotic tissue from the throat probably contains the organisms of Vincent's angina. It is poor judgment, however, to regard any mass in the neck in the region of the lymph nodes as a local disease until one has definitely excluded a primary neoplasm of the respiratory or of the upper digestive tract of which the mass in the neck is a metastasis. The absence of ulceration of the tonsil, of invasion of adjacent tissues and of a tumor nodule on the surface of the tonsil leads to many errors in the diagnosis of the nature of the enlargement. In fact, if the sarcoma is small and a cervical metastasis is large, the tonsil may be regarded as normal and the mass in the neck as a primary tumor.

A more subtle cause of diagnostic error results from the similar appearance of sarcoma and tonsil tissue. With the unaided eye one tissue cannot be distinguished from the other. The boundaries of a sarcoma cannot, therefore, be determined accurately by physical examination. This resemblance of the two tissues plays a critical role in the selection of the site of removal of a biopsy specimen, in the distribution of radon seeds and in the determination of therapeutic destruction of the sarcoma.

Sarcoma of the tonsil cannot be distinguished by physical examination from the other types of neoplasm of the tonsil, namely, squamous cell carcinoma, lympho-epithelioma and transitional cell carcinoma. The clinical pictures presented by all types of malignant growths may be identical. Identification of the nature of a patient's tumor, which has been discussed by New and associates,[2] depends on the microscopic examination of an adequate biopsy specimen. The difference in microscopic appearance of the various types of malignant growths in the tonsil is not always as great as the names suggest, and tumors of borderline type are not infrequent.

With all sarcomas, except the very large ones, an opinion regarding the size and position of the tumor in the tonsil must be based on information gained by the use of the laryngeal mirror, the pillar retractor and the palpating finger. The correctness of the diagnostic impressions formed by the use of these methods of physical examination can be tested by the removal and examination of biopsy specimens from the suspected areas.

A diagnosis is incomplete which does not include a determination of the location of all metastases. To secure this knowledge of the disease, a general physical and a roentgenographic examination of the chest are required. Before any treatment is planned, the abdomen should be examined with special care in order to exclude the presence of abdominal metastases. A roentgenogram should also be taken of the skeleton or any part of the body which has felt different since the onset of symptoms referable to the throat or the appearance of a mass in the neck.

All should be warned of the pitfalls in diagnosis of a small sarcoma resulting from the absence of evidence of malignancy in a biopsy specimen. In 5 of the 7 cases which I have encountered the first biopsy specimen did not contain neoplastic tissue, although the disease was sufficiently advanced to arouse a suspicion of the presence of a malignant growth in the mind of the referring physician. Postponement of treatment or ill advised operation, both dangerous to the patient, may result from the acceptance of a negative report as proof of the absence of a malignant process.

The absence of malignant tissue from the biopsy specimen may be due to the tendency of the operator to remove tissue from the part of the tonsil which is most

accessible (the middle third) rather than from the part which looks unnatural. When a biopsy specimen is excised, it is not wise to assume that the entire tonsil has been replaced by sarcoma or to forget that some sarcomas arise in the depths of the tonsil and are covered by a layer of normal tissue.

TREATMENT

Sarcoma of the tonsil and the regional metastases are treated with roentgen rays or radium or with a combination of the two. Roentgen ray treatment of sarcoma of the tonsil consists in the administration of daily doses of roentgen rays of moderately short effective wavelength and of a fraction of the amount which reddens the skin. The roentgen rays are delivered to the tonsil through an intraoral cone (fig. 4) and through the skin of the neck overlying the tonsil. The treatments are continued until the tonsil is covered by a diphtheritic membrane. An amount of roentgen radiation which blisters the skin may be necessary to produce membrane on the lower pole of the tonsil.

Students of therapeutic radiology [5] accept the formation of membrane as indispensable for local cure, but they may produce it by different roentgenologic technics. They use singly or in combination radon implants, teleradium and different doses of roentgen rays.

The implantation of radon seeds may be the sole method of treating sarcoma involving the tonsil. Radon, the gaseous disintegration product of radium, confined in tiny lengths of capillary gold tubing with a wall 0.3 mm. thick, may be permanently placed at intervals 1 cm. apart in the sarcoma and the surrounding tonsil. Seeds may also be used to supplement a smaller dose of roentgen rays than that which causes blistering of the skin. The combined method of irradiation offers the advantages of the diffuse radiation effect of roentgen rays and the more intense local radiation effect of radon seeds. Radon seeds have some advantages over radium needles. Seeds are easier to place accurately, more likely to stay in position and more easily obtained than are radium needles. There is no essential difference in the effect of the two on malignant tissue.

Metastases to the lymph nodes of the neck are treated with daily fractional doses of roentgen rays. The roentgen radiation is supplemented with gamma radiation from radium needles if the masses persist. The great probability of cervical metastases and the poor prognosis when they appear make prophylactic treatment of the neck with roentgen rays a logical procedure. Roentgen radiation may be given for relief of symptoms produced by thoracic, abdominal and skeletal metastases.

The well known radiosensitivity of sarcoma should not lead one to believe that sarcoma of the tonsil can always be destroyed by a small dose of radiation. It is true that some sarcomas shrink with amazing rapidity (fig. 3), but others require the maximum dose of radiation given to other types of malignant growths of the tonsil.

PROGNOSIS

The prognosis of sarcoma of the tonsil is poor and should be guarded. The immediate effect of treatment is gratifying to the physician and encouraging to the patient, but the final result is often disappointing. Many patients die of distant metastases months after control of the local disease. The 7 patients whom I have seen all died of distant metastases (table).

The probability of cure is not great if cervical metastases are present before treatment. The absence of signs of sarcoma in the tonsil and neck during the weeks or months following treatment may create in the examiner's mind a false impression

5. Coutard, H.: Principles of X-Ray Therapy of Malignant Diseases, Lancet **2**:1-8, 1934.

regarding control of the disease and a natural tendency to dismiss as unrelated to sarcoma vague pains and abdominal symptoms, particularly when the roentgenograms of the chest gives no evidence of metastasis. The disappearance of the primary tumor should not be confused with cure of the disease. The disease may consist of thoracic, abdominal and skeletal metastases, in addition to the primary neoplasm and the regional metastases.

A neoplasm arising from a flat surface, such as the cheek, is believed to have been destroyed when the tumor mass and the surrounding induration disappear. These criteria of control are not as reliable for sarcoma of the tonsil, a tissue which is by nature a mass. Reduction of a large tumor to the size of an average tonsil is not proof of the destruction of a sarcoma. The final opinion regarding control of the primary neoplasm must be based on the absence of evidence of malignancy in multiple biopsy specimens excised from various parts of the tonsil.

SUMMARY

A study of 7 cases of sarcoma of the tonsil has resulted in the following impressions:

Sarcoma of the tonsil is not a common tumor. It is not recognized early.

Sarcoma of the tonsil should be distinguished from squamous cell carcinoma of the anterior faucial pillar, the commonest malignant tumor in the region of the tonsil.

A large mass below the angle of the jaw is a common and conspicuous sign of sarcoma of the tonsil. A roentgenogram of the chest (negative for metastases) is not a reliable guide to the absence of abdominal metastasis.

Sarcoma tissue looks like tonsil tissue. Failure to note malignant growth at biopsy is meaningless; repeated biopsies may be needed for correct diagnosis.

A final opinion regarding the destruction of the sarcoma in the tonsil should be based on the absence of evidence of malignancy in specimens from different parts of the tonsil.

The sarcoma involving the tonsil and the cervical lymph nodes is destroyed by radiation, but the prognosis is poor because many patients later die of distant metastases.

158 Maplewood Avenue.

PRELIMINARY VOICE TRAINING FOR LARYNGECTOMY

JULIUS W. McCALL, M.D.
CLEVELAND

In no other organ of the body does carcinoma yield so successfully to early diagnosis and surgical treatment as in the larynx. Thomson,[2] in reviewing the history of carcinoma of the larynx, said:

> When we recall that fifty years ago Morell MacKenzie was obliged to say that, for cancer of the larynx, "the only possible termination is death," we may rejoice in realizing to-day that with laryngofissure we can effect lasting cures in over 80 per cent of cases, that there should be no operative mortality, that surgery (laryngofissure and laryngectomy) will cure practically all cases of intrinsic cancer and that, in skilled and careful hands, they are now well-established as safe and justifiable procedures.

The decision between laryngofissure and laryngectomy is not always easy, but if laryngeal surgeons would remember not to compromise with carcinoma, many more patients with cancer of the larynx would survive. Total laryngectomy does not offer technical difficulties, but if the surgeon is not willing to devote much time, effort and thought to patients who undergo this operation, to their social and psychologic problems and to their rehabilitation, he should not undertake their management.

With the improvement in results of surgical treatment of cancer of the larynx, the major problems still to be solved are education of the public with regard to the curability of this type of cancer and rehabilitation of the patient after laryngectomy. All recent authors have stressed the fact that the majority of patients with cancer of the larynx do not consult a specialist until the condition is far advanced. I regret that my own series illustrates this. The successful results of laryngectomy in patients with early carcinoma and the deplorable failure of patients to see a physician in time to avail themselves of the benefits of treatment prove that much remains to be done in educating the public that persistent hoarseness is a danger signal and should demand immediate examination by a qualified specialist. Public health authorities, organizations devoted to control of cancer and the medical profession should combine forces to work toward this end.

When this is done, the problem of rehabilitation of the laryngectomized patient will become increasingly important. The advance of modern surgery has brought many refinements in the preoperative and postoperative care of the patient with cancer of the larynx. The laryngeal surgeon's responsibility should now extend beyond the operative care and should provide systematic training to insure a speaking voice after operation, for this is of prime importance to the patient. In the past, the greatest objection to total laryngectomy on the part of both surgeon and patient was loss of the speaking voice. With preliminary voice training prior to operation this disability now can be overcome.

In the series of cases in which I have performed laryngectomy, the problem of training the patient in the development of the esophageal voice has received special attention. The method used has been so successful that I feel it should be more widely employed in the treatment of patients with carcinoma of the larynx.

Read at the annual meeting of the Eastern Section of the American Laryngological, Rhinological and Otological Society, Inc., Hartford, Conn., Jan. 15, 1943.

1. Footnote deleted by the author.

2. Thomson, St.C.: History of Cancer of the Larynx, J. Laryng. & Otol. **54**:61-87, 1939.

THE ESOPHAGEAL VOICE

The esophageal voice has been widely studied, but from a review of the literature it is evident that the idea of preoperative training in preparation for its development is new. Heretofore, voice training has been delayed until after the larynx was removed. If a small part of the time and effort expended is devoted to training patients before operation, they can be assured of a speaking voice. Orton [3] said that "every laryngectomized patient can talk with an esophageal voice if he makes up his mind to it." With this I agree, but the procedure is greatly simplified if training is begun before laryngectomy.

Czermak,[4] in 1859, recorded the first case of esophageal voice. Störck,[5] with Gersuny, reported a case in which such a voice was produced after total laryngectomy; Seiler [6] also described an early case. A patient operated on by Mikulicz was referred to Gottstein [7] for voice training, with brilliant results. Not only did he learn to speak, but his voice was capable of such modulation that he could sing within the range of almost an octave. In 1908 Gutzmann [8] reported 25 cases in which intelligible voices followed laryngectomy.

Morrison [9] stated:

> It seems essential that surgeons who perform total laryngectomy . . . should attempt to aid these voiceless persons in securing a useful pseudovoice. Such patients will not be cut off in their social intercourse, nor will they be unable to earn a living because of their lack of intelligible speech.[9a]
>
> In the late postoperative period the laryngectomized patient is apt to suffer from mental depression because of the social and economic barriers imposed on him by total aphonia. The best cure for this depression is a well guided attempt to learn to produce the pseudovoice.[9b]

My interest in the esophageal voice was stimulated by one of the patients, Carl Lange. By sheer determination and practice he has developed an excellent speaking voice.

Psychologic Preparation of the Patient.—When a positive diagnosis of carcinoma following biopsy reveals the necessity of total laryngectomy, both patient and surgeon are anxious to have the operation over. The surgeon's concern over the outcome of the operation, for the time being, is likely to overshadow the thought of the patient's later life. This concern is commendable, but if the patient survives, the ability to converse with those around him is almost vital; so it is important to give some thought to his later existence. If a few days of training before operation mean the difference between a good speaking voice and the alternative of his using some mechanical device, the patient will be much better off if the operation is delayed until this training is given. The surgeon has only to compare the happiness of the laryngectomized patient with an esophageal voice and that of one bereft of speech to realize what this means. The training period is short—a week to ten days—and does not materially affect the chance of survival. The foremost thoughts in the mind of a patient about to undergo laryngectomy are: "Will I live?" and "Will I talk?" It is now possible to assure such a patient that if he lives he will talk.

Fortunate is the surgeon who has one or more patients who have developed a good esophageal voice. It is of inestimable value to the patient contemplating

---

3. Orton, H. B.: Review of Diseases of the Larynx, Laryngoscope **50**:8-163, 1940.
4. Czermak, cited by Morrison.[9a]
5. Störck, cited by Morrison.[9a]
6. Seiler, cited by Morrison.[9a]
7. Gottstein, cited by Morrison.[9a]
8. Gutzmann, H., cited by Morrison.[9a]
9. Morrison, W.: (*a*) Production of Voice and Speech Following Total Laryngectomy, Arch. Otolaryng. **14**:413-431 (Oct.) 1931. (*b*) Morrison, W.: Physical Rehabilitation of the Laryngectomized Patient, ibid. **34**:1101-1112 (Dec.) 1941.

laryngectomy to see and talk to some one who has had the operation and has developed an esophageal voice. Persons who have accomplished this inspire confidence, improve morale and stimulate hope in the one who is about to undergo this ordeal. A patient with an esophageal voice is far more successful in "selling" the operation than is the surgeon. One patient in my series said: "When I found out what I had and what should be done, I decided to go home and use a gun, but after seeing and talking with C. L. I decided to have my throat cut instead."

Mechanics of Voice Production.—To quote Negus:[10]

> Voice is generally understood to refer to the production of sound by means of the pulmonary apparatus. There are many means by which an animal may throw the air in its neighborhood into sonorous vibrations, either by voluntary or involuntary movements of some part of its body. In a wide sense voice may be defined as the purposive production of sound for purposes of communication. A narrower, but more generally accepted view, limits the sound to one made by means of the pulmonary air tract.

Jackson[11] stated:

> In developing phonation, nature seems simply to have made use of the phylogenetically earliest primary valvular mud-excluding laryngeal function, using the opening and closing mechanism in opposition to each other.

Voice Production in the Laryngectomized Patient.—Dr. Normand Hoerr, professor of anatomy, Western Reserve University, and I made fluoroscopic studies of our laryngectomized patients who have developed an esophageal voice. The examinations were made from five weeks to six years after the operation.

All these patients have developed an esophageal voice good enough for them to be easily understood over the telephone. With the use of a Patterson B screen and with the tube set at 54 kilovolts and 6 milliamperes, we examined the pharynx, both with and without the aid of contrast material, during rest, during swallowing and in phonation.

The contrast mediums used were barium sulfate, dry or suspended in water, and barium sulfate rubbed up in butter and served on a dry soda biscuit. The last mixture is rather difficult to swallow, but as the butter softens, it causes the barium sulfate to stick to the mucous membrane of the pharynx. Thus, even though the pharynx is practically empty, the walls can be well outlined, and the medium offers no hindrance to the production of sounds. We observed the pharynx usually in left oblique and lateral positions, both while the patient was talking and while he was making vowel sounds only. Most patients with carcinoma of the larynx have a distinctly hoarse voice. In nearly all of them there is a distinct and easily recognizable difference, as seen under the fluoroscope, in the degree of constriction with their broad a and long o diphthong sounds.

After operation (as seen in the fluoroscope) the hyoid bone of the laryngectomized patient is about 1 cm. lower than normal. The base of the tongue, therefore, has dropped down somewhat, and the epiglottis is tilted posteriorly more than normal. The vallecula is larger than before operation, as a result probably of the backward tilting of the epiglottis.

The anterior wall of the pharynx has been completely sutured at operation; that is, those portions of the middle and inferior constrictor muscles of the pharynx which originally had their origins from the thyroid and cricoid cartilages have been sutured to their counterparts on the opposite side.

---

10. Negus, V. E.: Mechanism of the Larynx, St. Louis, C. V. Mosby Company, 1931.
11. Jackson, C.: Myasthenia Laryngis, Arch. Otolaryng. **32**:434-463 (Sept.) 1940.

Below the epiglottis, the anterior wall of the pharynx bulges forward or even pouches out. This outpouching is subject to considerable individual variation. There is another outpouching of the anterior wall of the pharynx at about the level of the seventh cervical vertebra, which also varies greatly. These pouches or bulges have an anteriorly convex wall, which is smoothly continuous with the rest of the pharynx. They are never so abrupt as to resemble diverticula. My opinion is that they do not in any way represent weaknesses in the pharyngeal wall but, rather, indicate an adaptation of the altered pharynx to somewhat enlarged surroundings, after removal of the larynx.

The pharynx, as a whole, is wider anteroposteriorly than normal. The normal pharynx may be completely flattened anteroposteriorly in a state of rest. The pharynx of the laryngectomized patient, on the other hand, never collapses, even in a state of rest. This may be due to the almost constant swallowing of air preparatory to esophageal regurgitation and voice production. In most patients, the pharynx, as seen on the fluorescent screen, remains open at least 5 mm.

At about the level of the sixth cervical vertebra, or the former level of the cricoid cartilage, there is a definite narrowing of the pharynx. I consider this narrowing to be due to the cricopharyngeus muscle, or the lower portion of the inferior constrictor muscle of the pharynx. During the phonation of vowel sounds, this portion of the pharynx becomes greatly constricted. If the patient attempts to produce a tone of high pitch, or if he makes the long e sound, the lumen of the pharynx (as seen from the side) may disappear completely for a fraction of a second, or even more than a second. In some patients the constriction is local, involving not more than 1 cm. of the length of the pharynx. In 2 of the patients the constriction involved 2 to 3 cm. of the length of the pharynx. In other words, the region of greatest constriction was usually not over 1 cm. in length, but might be as long as 3 cm. In 3 of the patients the constriction looked like a distinctly pointed backward projection of the anterior wall of the pharynx.

The constriction, however, consists not only of a posterior movement of the anterior wall of the pharynx but also of a forward movement of the posterior wall. In other words, the constriction is a sphincter-like action of the pharyngeal muscles, particularly of the cricopharyngeus muscle. In 2 of the patients there was considerable forward movement of the posterior pharyngeal wall. In 5 patients, however, the most noticeable feature of the constricting action was the posterior projection of a small bit of the anterior pharyngeal wall.

There was no special constriction of the pharynx at the aforementioned level during swallowing. In other words, the constriction was visible only on phonation. Patients differ in the ease with which they can retain swallowed air in the esophagus. Sometimes there is a cricopharyngeal narrowing just after air has been swallowed, which may also aid in the momentary retention of air.

I have come to the conclusion, therefore, that the vowel sounds may be produced by a vibration of the pharyngeal wall at the cricopharyngeal constriction. In a few patients (and these are the best speakers) this constriction, as seen from the side in the fluoroscope, is so local, and so extreme, as to remind one of the approximation of the vocal cords as seen with the laryngeal mirror.

Fluoroscopic studies in our series of cases confirmed the earlier observations by Negus and Morrison that the narrowed cricopharyngeal space is the site of sound production after laryngectomy. The cricopharyngeal space makes the esophagoscopist's work more difficult, but it is a boon to the surgeon and patient after laryngectomy in the development of an esophageal voice. To students of this problem, the work of Negus, Morrison and Jackson on the cricopharyngeal space should be fundamental.

Preoperative Training.—The surgeon should provide systematic training for the recovery of speech after laryngectomy. The esophageal voice is the result of timing and synchronization of movements of the lips, tongue and teeth with an adequate expulsion of air from the esophagus and pharynx. Definite narrowing of the cricopharyngeal space after laryngectomy creates sound vibrations when a sufficient volume of air is expelled from the esophagus. Therefore, development of an esophageal voice is dependent on the patient's ability to expel forcibly an adequate volume of air from the esophagus, that is, the act of belching. The laryngectomized patient's ability to develop an esophageal voice is absolutely dependent on his ability to master the technic of belching. If he does not learn this,

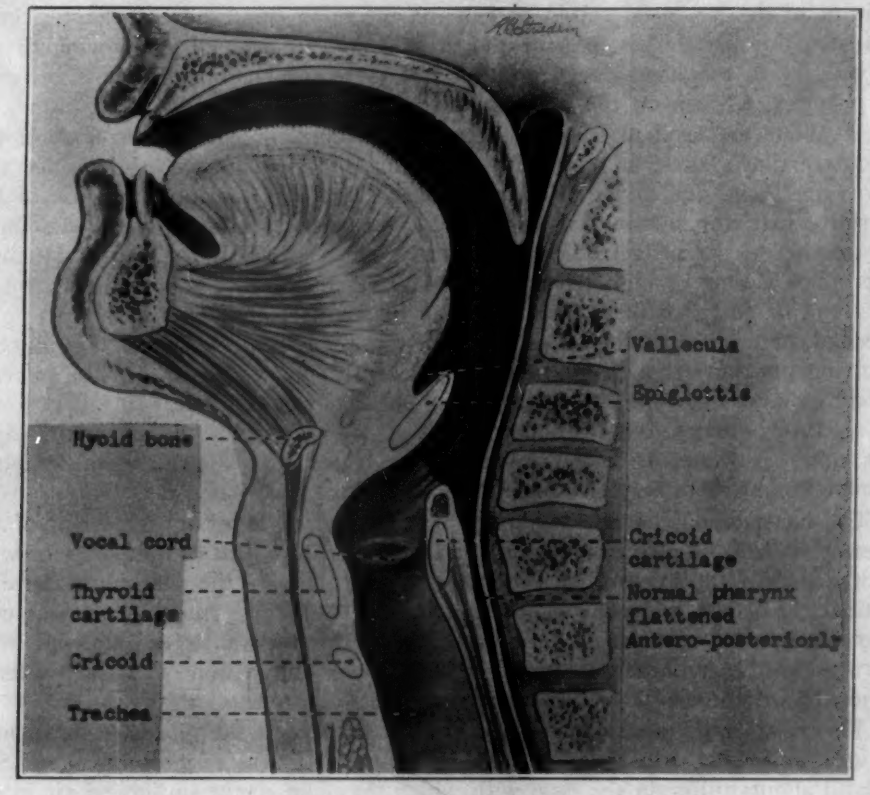


Fig. 1.—Sagittal section of the normal pharynx and larynx.

he will not develop an esophageal voice. It is much easier to learn to belch properly before the larynx is removed than afterward. If training is delayed until after operation, the check to respiration offered by the glottic valve is gone, and the patient will explode air from the trachea instead of the esophagus.

The belching effort is acquired easily by some patients; others find it difficult. By training patients in this effort for a week or ten days before laryngectomy, the greatest difficulty in the development of an esophageal voice is overcome. Most patients who must submit to laryngectomy desire a few days to arrange their affairs, and if this time is utilized in learning to belch, it will greatly shorten the postoperative period of training for development of the esophageal voice.

As an example, I shall cite briefly 2 cases in which operation was performed recently. A man aged 52, a painter, had voice training for ten days before total

laryngectomy. The feeding tube was removed six days after operation, and he had an immediate good speaking voice. A woman aged 61, a housewife, had voice training for five days before total laryngectomy. The feeding tube was removed on the tenth day. She has an excellent speaking voice and, to the best of my knowledge, has been talking continuously ever since, proving that the old saying, "A woman will talk," still holds good.

To aid the patient in learning to trap air in the esophagus, carbonated drinks are prescribed. The patient is told to inhale deeply and then cease active respiration while he eructates air from the stomach and esophagus.

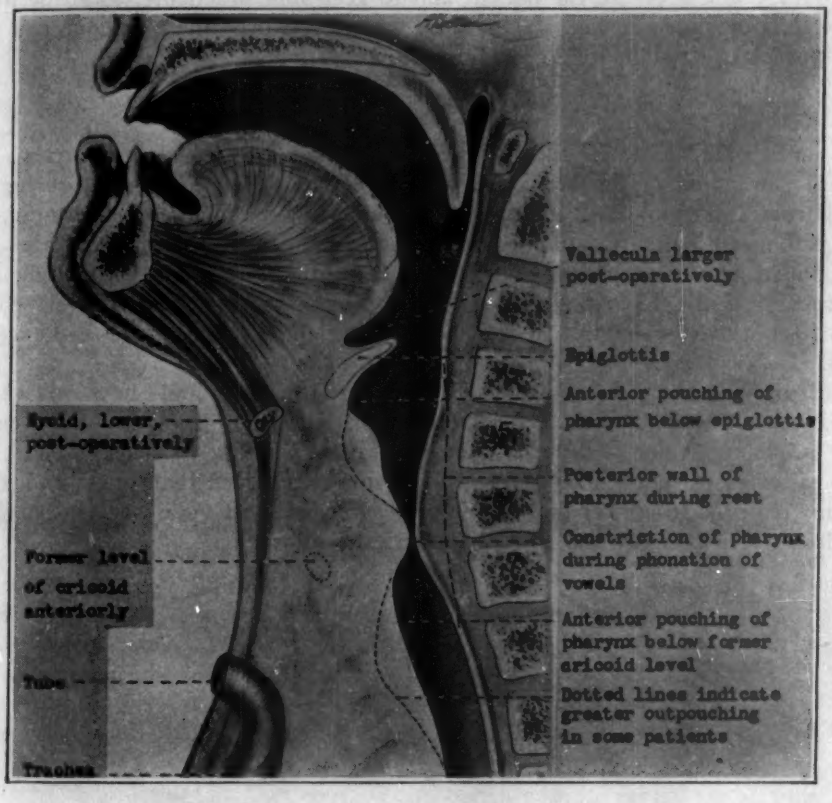

Vallecula larger post-operatively
Epiglottis
Anterior pouching of pharynx below epiglottis
Posterior wall of pharynx during rest
Constriction of pharynx during phonation of vowels
Anterior pouching of pharynx below former cricoid level
Dotted lines indicate greater outpouching in some patients
Hyoid, lower, post-operatively
Former level of cricoid anteriorly
Tube
Trachea

Fig. 2.—Fluoroscopic view of a laryngectomized patient during phonation.

Postoperative Training.—As soon as the feeding tube is removed, the patient is required to resume the belching effort. At first a great deal more stress is placed on having the patient belch than on having him try to form words. The words "O. K." and "Hello" seem to be the easiest to form. In the beginning, the patient is tempted to try to form sentences. This should be discouraged, for he should try one word at a time. When he has achieved the formation of single words, it is an easy matter to put phrases and sentences together.

The greater the cooperation and understanding between the surgeon and the patient in the preoperative and the postoperative training period, the better will be the results. The surgeon, in directing the voice training, should see the patient often, for there are many times when he and his family are discouraged.

Not only does the patient become discouraged, but he is likely to develop faults that are hard to eradicate, such as the employment of writing as a means of communication or the use of the whispered voice. The whispered voice is much more easily acquired after laryngectomy than is the esophageal voice. It is generally unsatisfactory, as it has no volume, and once the habit of using the whispered voice is acquired, the patient will cling to it, unless close supervision breaks him of it.

It is insisted that the patient devote at least two or three hours a day to the effort to talk. If he segregates himself from his family and friends, he is more likely to keep at practicing than if some one is around. It is well to enlist the sympathy and cooperation of the wife in this undertaking, for she can do much to encourage and reassure the patient and to keep him practicing, when he might otherwise be negligent. The ability to be understood over the telephone has been set as an arbitrary standard in judging the voice.

SUMMARY

A series of 32 cases in which laryngectomy was performed in a series of 38 cases of carcinoma of the larynx at St. Luke's Hospital, Cleveland, during the period from 1936 into 1942 illustrates the value of preliminary voice training for laryngectomized patients. Of 19 patients who had no preliminary training in development of the esophageal voice, only 3 acquired the ability to speak after operation. Of the 13 patients who had preliminary training, all developed the ability to speak well enough to be heard over the telephone, and several had excellent voices.

Patients who are to be subjected to total laryngectomy should have voice training before, not after, operation, for the following reasons: First, the development of an esophageal voice is dependent on the patient's ability to belch; second, it is much easier to learn to belch before the larynx is removed than after, and, third, the time required to master the technic of belching is short (one week to ten days) and does not jeopardize the patient's chance of survival.

610 Rose Building.

MECHANISM OF PHONATION DEMONSTRATED BY PLANIGRAPHY OF THE LARYNX

BRUNO L. GRIESMAN, M.D.
NEW YORK

I have used planigraphy in the study of the physiology of the larynx during phonation. Phonation starts with breathing. Most of the laryngeal sounds for the purpose of speaking or singing are produced during expiration. Breathing during speaking differs in many ways from breathing during rest. When at rest one inhales and exhales a small amount of air, about 500 to 600 cc., but for the purpose of speaking one must inhale about 1,500 to 2,400 cc. of air or more. During rest the periods of inhalation and exhalation are equal. During speech the period of exhalation is much longer than that of inhalation. Inhalation should be rapid and noiseless. Exhalation is audible as an aspirated sound. As the inspiration becomes deeper and inaudible, the vocal cords come to lie almost flush against the lateral wall of the larynx. The chink of the glottis becomes almost the same size as the lumen of the trachea. During expiration, however, there are narrowing of the chink and adduction of the vocal cords, ventricles and ventricular folds toward the midline. This approximation of the false and true cords produces resistance to the expiratory current of air, and therefore a friction noise.

During expiration without tone production there is a wide glottic chink, through which air escapes easily. In order that a tone may be produced the vocal cords are drawn close together in the position of phonation. There remains always a very small phonatory gap between the approximated vocal cords. This gap is necessary because there must be some room for the vibrations of the vocal cords.

The approximated vocal cords are set in vibration by the air pressure from below. The obstructed air current escapes in puffs during the opening phase of the vibration and is interrupted by the closing phase.

The vibrating vocal cords interrupt the constant flow of the air current and generate condensation and rarefaction of air, i. e., a sound wave. The pitch of this sound wave is determined by the frequency of the interruptions a second. The sound originating at the vocal cords consists of a fundamental tone and numerous harmonically related overtones. The changing shapes and dimensions of the resonating cavities, particularly the air column spaces above the vocal cords, such as the supraglottic space, the pharynx, the mouth and lips, the nasal cavities and the nasopharynx, diminish or amplify certain overtones and give the voice its characteristic qualities and human peculiarity.

Figure 1 demonstrates four phases of sound production. All planigraphs were made at the same depth of 2 cm. behind the Adam's apple. *A* shows the larynx sounding the vowel *a* at a frequency of 435 cycles; *B*, the vowel *u* at the same frequency; *C*, the vowel *u* one octave lower, at a frequency of about 218 cycles, and *D*, the vowel *u* one octave higher, at a frequency of 870 cycles.

Read at the meeting of the New York Academy of Medicine, Section of Otolaryngology, May 20, 1942.

From the Department of Otolaryngology, Presbyterian Hospital, and Vanderbilt Clinic, Columbia University College of Physicians and Surgeons.

A change in the vowel from *a* to *u*, even when sung at the same frequency, causes a slight lowering of the entire larynx, which seems to be necessary for the *u* sound. Moreover, one recognizes a decided gross movement downward of the entire larynx during the production of the low octave sound and a reverse movement upward during the production of the higher note.

These gross movements of the larynx for low and high tones are necessary, for two reasons: On the one hand, the change of the resonating air columns

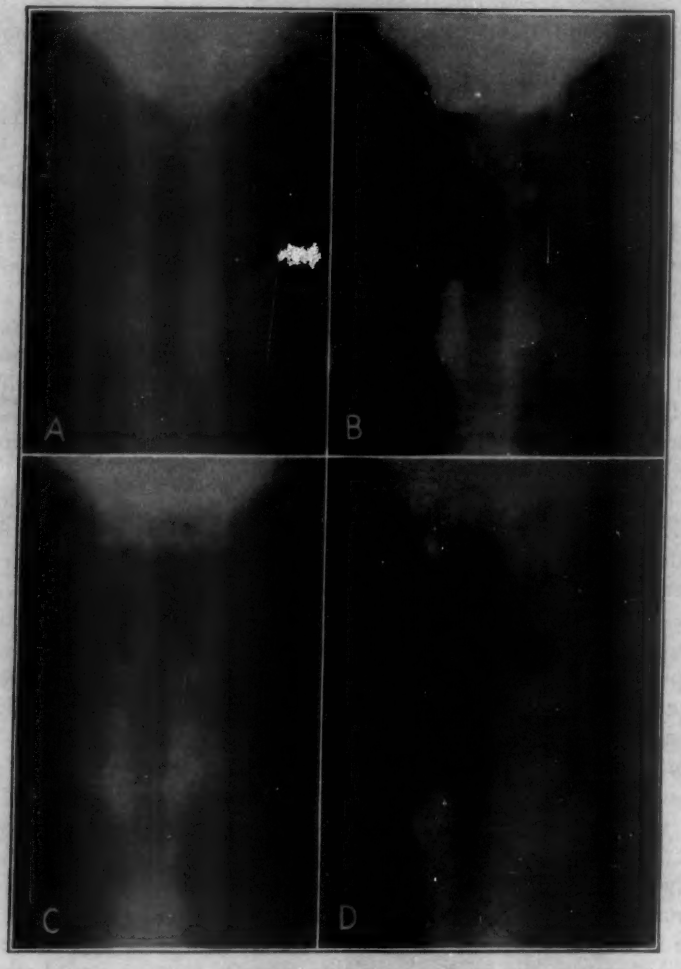


Fig. 1.—Four phases of sound production. All the planigraphs were taken 2 cm. behind the Adam's apple. *A*, tone of 435 cycles sung on the vowel *a*; *B*, tone of 435 cycles sung on the vowel *u*; *C*, tone of 218 cycles sung on the vowel *u*, and *D*, tone of 870 cycles sung on the vowel *u*.

in length and width aids the vibrations of the vocal cords, and thus regulates the pitch. A well known physical experiment uses a fishtail burner through which the breath is blown to set in vibration the air column in a tube. If the tube is raised and lowered rapidly, the pitch will vary as the length of the air column changes. The shorter the column, the higher the pitch.

On the other hand, these gross movements of the larynx have a distinct influence on the tension of the vocal cords. The vocal cords can be shortened both actively and passively, but they can be elongated only passively. The sternothyroid and the hyothyroid muscle both insert into the oblique line of the thyroid cartilage. The short fibers of one are opposite the long fibers of the other.

In lowering of the larynx, the tracheal pull downward rotates the cricoid plate forward, and the contraction of the sternothyroid muscle rotates the thyroid cartilage backward. These movements are accompanied by a foreshortening of the vocal cords because of approximation of their points of origin and insertion. The cricothyroid space increases. In elevation of the larynx three important factors play a part: (1) the elevation of the hyoid bone, especially its posterior part, by the action of the anterior belly of the digastric muscle; (2) the forward pull of the hyoid bone by the action of the geniohyoid muscle, and (3) the contraction of the hyothyroid muscle. These three factors change the oblique line of the

Fig. 2.—Vocal cords of a bass singer (2 cm. behind the Adam's apple). *A* shows sagging of the vocal cords during production of a tone of 173 cycles; *B*, the larynx during production of a tone of 256 cycles.

thyroid cartilage into a more vertical one, and elongation of the vocal cords is produced. The cricothyroid space diminishes.

Raising of the larynx means elongation and tightening, and lowering means shortening and loosening, of the vocal cords, movements resulting from the activity of the muscles of the neck. This passive change in the length and tension of the vocal cords, due to the action of the muscles of the neck, can be augmented by: (1) contraction of the thyroarytenoid muscles, causing active shortening, and (2) contraction of the cricothyroid muscles, causing passive elongation.

Figure 2 shows how the vocal cords of a bass singer act during production of low tones. At the left the larynx is shown giving a tone of a frequency of 173 cycles, and at the right, it is giving a tone of a frequency of 256 cycles. Production of the lower tone is characterized by thick, sagging and slackened vocal cords. The walls of the glottis are long, and the infraglottic aspect is concave.

The vocal cords give the impression of heaviness. The ventricle, the ventricular folds and the pyriform sinuses can be clearly visualized. With the higher tone of the bass voice the vocal cords are more contracted, of lighter weight and thinner, with the inferior aspect rectangular, the ventricular folds more tensed and the supraglottic space wider. The ventricle has a slightly different shape, and the entire larynx seems elevated.

The sphincters of the larynx are arranged in two groups, the external and the internal.

In the beginning of evolutionary development, the external (pharyngolaryngeal) sphincter surrounded both the pharynx and the larynx like a girdle. This external sphincter is supplied by the superior laryngeal nerve. Even in man this nerve supplies the rest of the system, namely, the inferior pharyngeal constrictor muscle and the two cricothyroid muscles.

The internal sphincter, on the other hand, is highly developed in man. It differs from the external sphincter in that it does not envelop the pharynx. It is a muscle system which, inserting in the thyroid cartilage, encircles the arytenoid and the cricoid cartilage. The internal sphincter represents the intrinsic muscles of the larynx: the two lateral and the two posterior cricoarytenoid muscles, the two thyroarytenoid muscles and the single interarytenoid muscle.

The vocal muscle can contract in two directions—longitudinally and circularly, as a sphincter, in connection with its fellow on the other side. It seems that each function is the property of distinct muscle fibers and that the longitudinal contractility is stronger than the sphincter action of the vocal muscle and its associates.

The larynx can be closed at three levels: at the level of the true vocal cords; at the level of the ventricular folds, and at the level of the aditus laryngis, the aryepiglottic sphincter. As can be seen, the complete sphincter mechanism, the external and the internal sphincter, is under the control of the recurrent and the superior laryngeal nerve. This threefold safeguarding of the airway, through three sphincteric mechanisms, can be brought into action at once, contraction occurring at all the levels together or at each level separately, as an individual unit.

My planigraphs demonstrate that the primary function of the larynx as a sphincter valve plays also an important role secondarily in the production of voice.

I studied tone production at three successive steps in the scale of a tenor: the frequencies of 274 and 870 cycles and a high falsetto tone. At the lower chest note the entire larynx, the true and false cords, the thyroid cartilage, the conus elasticus and the laryngeal vestibule appeared blurred, being set in forced vibration because of the powerful voice and the intensity of the sound. These forced vibrations caused reenforcement of the glottal note. The blurring between the vocal cords closed the gap and indicated complete approximation of the cords during vibration.

The frequency of 870 cycles, already a high tone, shows much less forced vibration. Only the upper third of the glottic wall comes into contact. Still clearer and sharper appears the configuration of the vocal cords during the production of the high falsetto tone (fig. 3). The cords look like small birds picking with their beaks against one another. There is no blurring of the surrounding tissues. Only the upper free margins of the vocal cords are vibrating.

Before the development of planigraphy it was difficult to get an idea of the lower aspect of the vibrating vocal cords and the contours of the resonating tubes.

Many investigators used dead larynxes for their observations. Merkel [1] experimented with such a larynx in 1863. He used a pipe bowl and inserted into one opening a short tube connected with the trachea of a dead larynx. From the other opening of the pipe bowl a longer tube was led to a mouth piece. Using his own breath, he observed the movements of the vocal cords. A string pulled through the cricoid plate and tied to the breathing tube served to fix the larynx. Another string was drawn through the thyroid cartilage above the insertion of the vocal cords. By pulling on this string the vocal cords could be stretched and elongated. Merkel could produce nearly the entire range of tones with his dead larynx—chest tones as well as falsetto tones. In his book he included a diagram of the canal of the glottis during production of falsetto tones. There is a striking similarity between the living and the dead vocal cords during the production of such tones. Nearly eighty years was required for the establishment of Merkel's observations.

Fig. 3.—Vocal cords of a tenor singing a falsetto tone (2 cm. behind the Adam's apple). The vocal cords look like small birds picking with their beaks against one another.

Figure 4 is the planigraphs of a baritone singing the vowal e (as in elect) at the frequency of 218 cycles (*A*) and, again, one octave higher, at a frequency of 435 cycles (*B*). It shows the phonatory changes in the dimensions and the differences in the shape of the supraglottic air space.

These changes in the supraglottic and the infraglottic column of air are of substantial aid in the production of vibrations of the vocal cords. A wave sent out from the vocal cords may be reflected back to reach the vocal cords at the same phase, and so increase the violence of vibration. Some well trained singers use their vocal cords without much help from variations in length of the supraglottic or the infraglottic air column.

1. Merkel, C. L.: Anatomie und Physiologie des menschlichen Stimm- und Sprach-Organs, Leipzig, A. Abel, 1863, p. 514.

The supraglottic and infraglottic columns of air are set in vibration by the vocal cords. There is a considerable analogy between laryngeal phonation and tone production by the vibration of the lips in playing an air column instrument, for example, a bugle or a cornet.[2] The mouth piece of a bugle shows a narrowing above its opening. The space of a mouth piece is never purely cylindric, but is always conic. The reason for this is the favorable mechanical effect on the vibrations of the lips of the congestion of the sound waves below the narrowing. The mouth piece of an air column instrument and the supraglottic air space of the larynx look slightly similar. It is easy to understand that, in analogy with the mouth piece of the instrument, the supraglottic air space of the larynx increases the vibration of the vocal cords and acts as a resonator, reenforcing or suppressing certain partial tones. The supraglottic space appears to be chiefly narrowed by

Fig. 4.—Planigraphs of a baritone (2 cm. behind the Adam's apple). *A*, frequency of 218 cycles, and *B*, frequency of 435 cycles.

sphincteric action during production of low tones. The rigidity of the supraglottic space increases with the elevation of pitch.

Figure 5 demonstrates four steps in the production of tones in the ascending scale of a mezzo-soprano: *A*, a low chest tone of 435 cycles; *B*, a middle tone of 870 cycles (the octave); *C*, a falsetto tone, and *D*, an extremely high falsetto tone. The different aspects of the infraglottic space, the change from the dome-shaped to the rectangular contour, can clearly be seen as a sign of higher muscle tension.

As the pitch is raised, two things take place: the thyroarytenoid muscle contracts more and more powerfully, so that the cords are firmer, and the cricothyroid

2. Jackson, C.: Myasthenia Laryngis: Observations on the Larynx as an Air Column Instrument, Arch. Otolaryng. **32**:434 (Sept.) 1940.

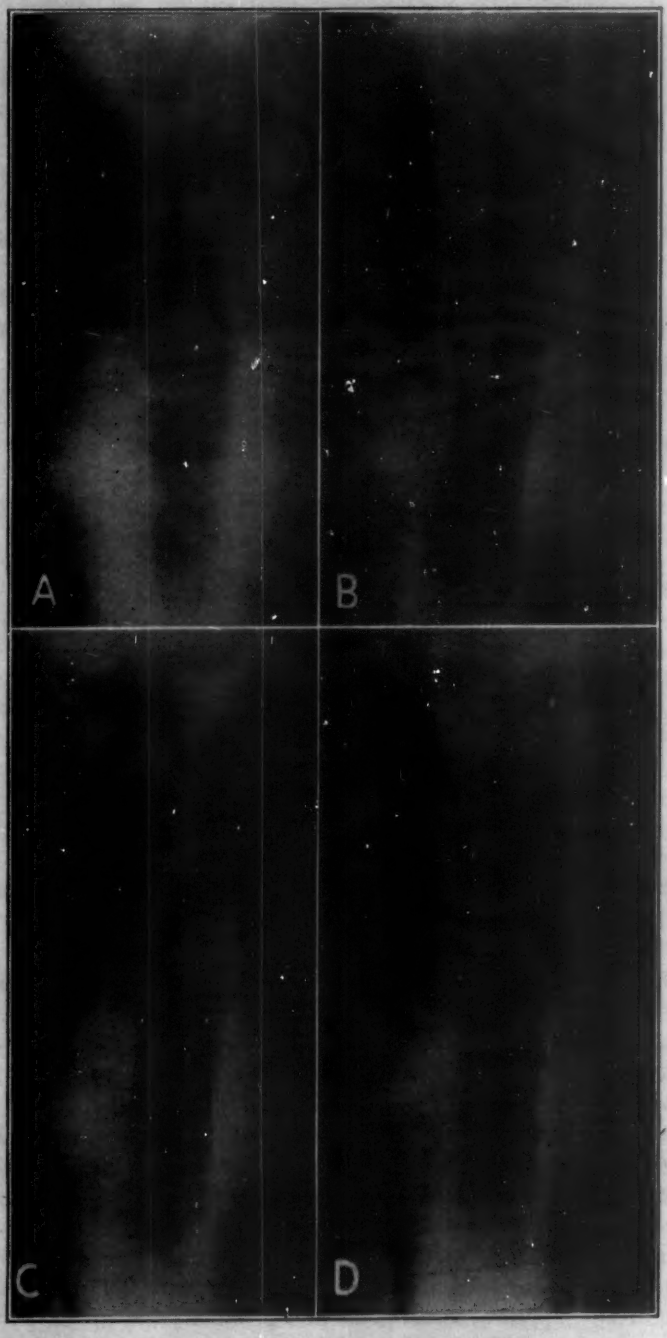


Fig. 5.—Planigraphs of a mezzo-soprano singing the ascending scale (taken 2 cm. behind the Adam's apple). *A*, chest tone of 435 cycles; *B*, tone of 870 cycles; *C*, falsetto tone, and *D*, extremely high falsetto tone.

and the posterior cricoarytenoid muscles contract accordingly, so that the cords are longer. Therefore, as the singer ascends the scale, the closely approximated vocal cords become more and more stretched, lengthened, thinner and tauter. There finally comes a point at which any further active contraction of the passively elongated vocal muscle becomes impossible. This is the point at which one can hear a break in an untrained singer's voice in going farther up the scale. At this point the mechanism of voice production changes from the so-called normal, or chest, mechanism into the mechanism for falsetto tones for further raising of the pitch.

The powerfully contracted vocal muscle relaxes suddenly and gives up its antagonistic action, so that the combined stretching effect of the cricothyroid muscle and its intrinsic and extrinsic associates can go on without resistance from within. During the production of higher tones only the free edges of the vocal

Fig. 6.—Larynx during whispering of the sound ã (as in "father").

cords, the vocal ligament and the innermost fibers of the vocal muscle, the aryvocalis muscle, are vibrating. Further elevation of pitch is accomplished by more powerful elongation of the vocal cords through the cricothyroid muscle and by elevation of the entire larynx through the muscles of the neck. Finally, there comes a point in ascending the scale at which further lengthening of the edges of the vocal cords is impossible. In order to reach the highest tones the sphincter mechanism must finally come into action.

Planigraphic examination of the larynx during production of such a very high tone shows that the larynx is in a state of spasm (fig. 5 *D*). The larynx is elevated to a maximum and appears compressed and strained; the air is exhausted; the supraglottic space is narrowed; the vocal cords have obtuse angles, and all the tissues of the larynx, including the false vocal cords and the aryepiglottic folds, are contracted like a sphincter. In contrast to this, however, with a high tone of 870 vibrations the supraglottic space is wide, well aired and extended with the vocal

cords rectangular (fig. 5 *B*). With the extremely high falsetto tone the vocal cords are in firm apposition. There is no phonatory gap, no blurring between them, visible. This planigraph for the very high tones is better understood when it is compared with the photograph of the vocal cords during production of these tones.[3] The planigraph (fig. 5 *D*) shows only a section through the approximated and dampened parts of the glottis. The very small vibrating portion of the cords is not demonstrated in this planigraph.

Figure 6 shows the condition of the larynx during whispering of the vowel *a* (as in "father"). It is known through laryngoscopic examination that a wide triangular gap is often present posteriorly during whispering. But such examination does not seem to show the real appearance of the larynx during whispering. No pyriform sinus or ventricle is visible. The ventricle does not give free play to the vibrating cords. One can see the narrow subglottic space as a sign of the decreased air pressure, the dome-shaped inferior aspect of the approximated vocal cords, with a clearly blurred gap, indicating that the vocal cords perform real vibrations. The supraglottic space is obliterated because of approximation of the ventricular folds and the aryepiglottic folds above them. There is only a small outlet for the escaping air. The sphincter girdle is contracted at all three levels, but this contraction is less pronounced than in the contraction of the same girdle for fixation of the thorax.

When I obtained the first picture for such a whispered vowel, I doubted whether I was at the correct depth; perhaps I was too far toward the front or toward the back. Therefore I examined another patient carefully and made two planigraphs at exactly the same distance of 2 cm. behind the Adam's apple. The first planigraph demonstrated humming, with the mouth closed, at a low frequency of 194 cycles, a loud "mm"; the second planigraph, whispering of the vowel *o*.

Comparison of the position of the mandible and clavicle in the two pictures revealed that during whispering the entire larynx was elevated. It appeared compressed from below and closed internally in the supraglottic region because of the approximation of the ventricular and the aryepiglottic folds. The hyoid bone itself was elevated, and the distance from the hyoid bone to the glottis was decreased.

These pictures for whispered sounds are instructive. They prove not only that the vocal cords are not at rest during whispering but that they are actually vibrating, and they show that not only the vocal cords, but the entire larynx, is in a condition of increased effort and strain. It may be an open question which is more harmful to the voice, shouting or whispering.

I studied also the tightening of the sphincteric girdle in a woman as a result of her holding a heavy weight in both hands, with the forearms stretched out. As compared with the sphincteric closure during whispering, there was a definite increase in muscular action during thoracic fixation. The pyriform sinuses were clearly visible. Respiration had completely ceased.

When a subject was made to press his hands together as forcibly as possible and produce a tone like that made during defecation, a pressed *ä*, the contraction of the sphincter group of muscles, with a small canal for escaping air, could be seen. At levels 1, 2 and 3 the two sides were approximated.

Spastic dysphonia is a voice disorder with a typical unphysiologic function of the sphincter girdle. It is a slight laryngeal spasm, with hyperactivity of the sphincter group of muscles, especially the sphincter of the false cords. Normally

3. Pressman, J. J.: Physiology of the Vocal Cords in Phonation and Respiration, Arch. Otolaryng. **35**:355 (March) 1942.

a speaker is capable of closing the true vocal cords independently of approximation of the false cords. But in the case of spastic dysphonia this ability has been lost. Approximation at levels 1 and 2 is simultaneous. In such a case of disturbed muscular stimulation the effectiveness of muscular contractions in other parts of the body, especially arm efforts, on the condition of the supraglottic space and the sphincter mechanism can be shown easily. The patient was asked to sing the same tone (*a*, at a frequency of 435 cycles) once as usual and then with his fists clenched. The narrowing of the laryngeal vestibule can be seen through the aryepiglottic sphincter (fig. 7). This experiment is the scientific proof of the effectiveness of the so-called pushing exercises for paretic voices introduced into the treatment of voice disorders by Froeschels.

It is not only for therapeutic purposes in cases of paretic weakness of the sphincter of the vocal cords that use can be made of this reflectory influence of

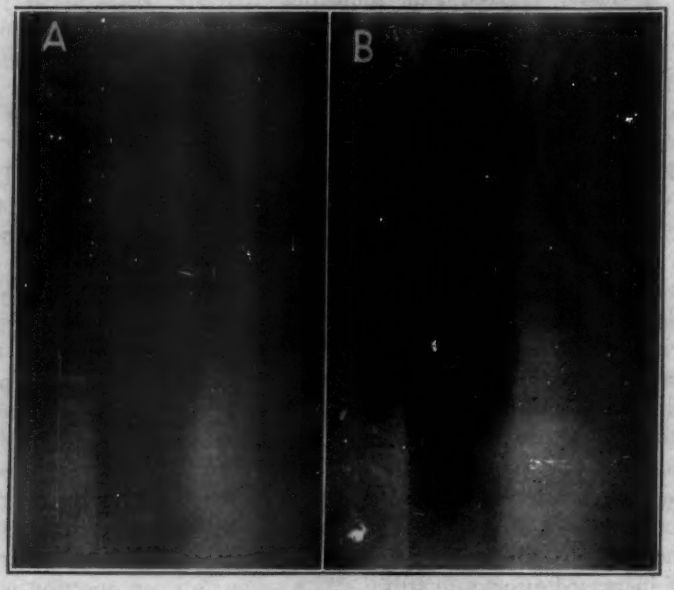


Fig. 7.—Planigraphs of a patient with spastic dysphonia (2 cm. behind the Adam's apple). *A*, tone of a frequency of 435 cycles; *B*, the same tone sung with the fists clenched.

arm efforts. One can frequently observe an orator utilizing this reaction, whether he knows it or not.

In the case of spastic dysphonia without phonation, when the hands are clasped, the tendency of the ventricular bands and of the vocal cords to approach and touch each other can be so strong as to create an air space between them which connects the two ventricles.

In the examination of patients with voice disorders it remains to be shown how the resonating cavities act, and not the vocal cords alone. To demonstrate this is one of the advantages of planigraphy over laryngoscopy and photography of the larynx.

Planigraphy of the larynx has advanced knowledge of the physiology of the sound-producing and sphincteric mechanisms. There are still many problems to be solved before definite rules for the functioning of a normal voice can be established.

1100 Park Avenue.

TREATMENT OF DYSPHAGIA FROM HERNIA THROUGH ESOPHAGEAL HIATUS IN DIAPHRAGM

PORTER P. VINSON, M.D.
RICHMOND, VA.

Herniation of an abdominal organ through the diaphragm into the thorax occurs frequently, and unless physicians are aware of the significance of symptoms produced by this abnormality, the nature of the disability may not be detected. Herniation through the diaphragm into the thorax may result from congenital maldevelopment or from rupture of a normal diaphragm by external trauma. In many instances a trivial congenital defect in the diaphragm that does not cause symptoms may be increased by the thoracic action of suction and positive intra-abdominal pressure so that later in life significant discomfort is produced. The purpose of this paper is not to discuss all types of diaphragmatic hernia, but to direct attention to two types in which dysphagia is often the predominating symptom.

In patients with hernia of one of these types, the esophageal opening in the diaphragm is larger than normal, and a portion of the stomach, usually the fundus, protrudes through the diaphragm alongside the esophagus, into the thorax. At first this abnormality may not produce symptoms, but because of negative intra-thoracic and positive intra-abdominal pressure more and more of the stomach is sucked and pushed into the thorax, so that eventually various symptoms occur. This type of hernia is designated as paraesophageal (fig., *A*).

The other kind is the so-called short esophageal hernia. In patients with hernia of this type the esophagus is usually about 2 inches (5 cm.) shorter than average (fig., *B*). The esophagus is not long enough, therefore, to reach the diaphragm, and in the process of growth a portion of the stomach develops within the thorax. A large or a small portion of the stomach may present above the diaphragm, but usually this type of hernia is about 2 inches (5 cm.) in diameter.

During the past six years I have observed 53 diaphragmatic hernias of the two types just described. Eleven were diagnosed as paraesophageal hernia, 3 being in men and 8 in women. Forty-two were diagnosed as congenital shortening of the esophagus with herniation of the stomach through the diaphragm, or hernia of the so-called short esophageal type. Of the latter group 18 were in men and 24 in women. All the patients were more than 40 years of age, the oldest being 83.

METHODS OF DIAGNOSIS

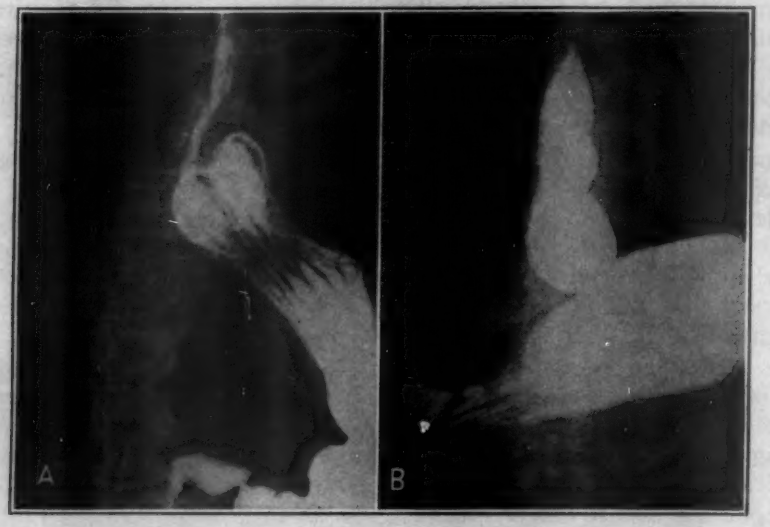
History.—On the basis of history alone the two types of hernia may be indistinguishable. In the majority of my patients difficulty in swallowing was the dominant discomfort, and it was more pronounced in those with short esophageal than in those with paraesophageal hernia.

In patients with the short esophageal type of diaphragmatic hernia dysphagia results from spasm or stricture at the junction of the esophagus and the hernial sac. In persons with hernia of the paraesophageal type it results from stricture in the lower portion of the esophagus, spasm at the cardia or pressure from the herniated portion of the stomach on the lower part of the esophagus. One fact that is difficult

From the Medical College of Virginia.

to explain is that in patients who have congenital shortening of the esophagus dysphagia seldom occurs before the age of 40 years, though the deformity has presumably been present since birth.

When obstruction results from spasm, dysphagia associated with hernia may be identical in character and severity with that accompanying other esophageal lesions. With hernia the difficulty in swallowing is usually intermittent, whereas with other types of esophageal obstruction it is more constant. Regurgitation of food is seldom experienced by patients with hernia, although fear of choking is constant, and dysphagia may be noted in swallowing any type of food, whether solid or liquid. The frequency of complete esophageal obstruction from a bolus of meat testifies to the intermittency of obstruction in the short esophageal type of diaphragmatic hernia. On numerous occasions I have had to remove such a foreign body with the aid of the esophagoscope. In 1 of my patients suffering from hernia of the short esophageal type, a bolus of meat which became lodged in the



A, paraesophageal diaphragmatic hernia; note the normal length of the esophagus, with herniation of the fundus of the stomach through the esophageal hiatus in the diaphragm, alongside the esophagus. *B*, short esophageal diaphragmatic hernia.

esophagus was forced into the stomach by the patient's drinking water. This was followed by profuse hemorrhage.

Ulceration may be present at the junction of the esophagus and the hernial sac, and this area may bleed. Recurring ulceration and healing may result in formation of cicatricial stricture. Hemorrhage may occur also in patients with paraesophageal hernia, the site of bleeding being the point at which the stomach is constricted by the diaphragm. When cicatricial stricture is present in diaphragmatic hernia, dysphagia is indistinguishable from benign stricture from other causes.

Another symptom of importance and one that is much less common and severe in patients with other esophageal lesions is distress from gaseous distention, especially after meals and when the patient is lying down at night. The degree of distress usually depends on the size of the hernial sac and is therefore more pronounced in patients with paraesophageal hernia. Sleeping in a semireclining position is often helpful in relieving the feeling of pressure and suffocation associated with lying flat in bed.

In 60 to 70 per cent of the patients who have cardiospasm, epigastric pain is an outstanding symptom. Although the cases of diaphragmatic hernia described here are too few to permit an accurate estimate of incidence of symptoms in terms of percentage, yet epigastric and substernal pain occurs with considerable frequency and is indistinguishable not only from the pain of cardiospasm but from that produced by gallstone colic or by disease of the coronary vessels of the heart. The type, severity and radiation of pain in all these diseases may be identical, and in a number of my patients coronary heart disease had been diagnosed previously.

Severe anemia may occur in patients who have either the short esophageal or the paraesophageal type of diaphragmatic hernia. Bleeding may be a sudden, severe hemorrhage or prolonged oozing from an area of ulceration. If the bleeding is of the latter variety, the cause of the anemia may be difficult to determine.

Roentgenoscopic Examination.—Roentgenoscopy is almost indispensable in the diagnosis of hernia through the esophageal hiatus in the diaphragm and in the differentiation of congenital shortening of the esophagus with hernia from paraesophageal hernia. Unless, however, hernia is suspected and the patient examined carefully, the lesion may be overlooked or the observations interpreted incorrectly.

I have observed patients who had been studied roentgenoscopically on several occasions and yet had not had the presence of a hernia detected, because the examination had been made with the patient in an erect position. Unless the patient is examined in the recumbent or the Trendelenburg position, the presence of hernia may not be apparent. If the patient is permitted to swallow a few mouthfuls of a fairly thick suspension of barium sulfate while in an erect position and then a mouthful or more of the same mixture while lying down, hernia, when present, will usually be detected and the type readily determined.

When cicatricial stricture has developed in a patient with hernia, the lumen of the esophagus may be narrowed sufficiently to prevent free ingestion of enough barium sulfate to permit satisfactory roentgenoscopic examination. In cases of this kind the stricture must be dilated and roentgenoscopy repeated.

Esophagoscopic Examination.—Direct visualization of the esophagus through an esophagoscope is a useful procedure in the diagnosis of diaphragmatic hernia. In patients who have congenital shortening of the esophagus with hernia, the junction of the esophagus and the stomach is found at a higher level than when the esophagus is of normal length. The presence of ulceration or stricture can be determined, and differentiation can be made from malignant obstruction.

Another diagnostic finding at esophagoscopic examination of a patient with this type of hernia is that as the esophagoscope is passed through the area of spasm or stricture at the junction of the esophagus and the stomach, gastric mucosa can be seen directly ahead of the examining tube even before the instrument enters the hernial sac. In a person with a normal esophagus or in a patient with cardiospasm or hernia of the paraesophageal type, the esophagus inclines toward the left and anteriorly, so that gastric mucosa is not seen until the tube has actually been introduced into the stomach.

DIFFERENTIAL DIAGNOSIS

Hernia through the esophageal hiatus in the diaphragm must be distinguished from various other esophageal lesions as well as from coronary heart disease and intra-abdominal lesions, especially chronic cholecystitis with or without stones. Inasmuch as symptoms from hernia are usually not pronounced until after the age

of 40 years, disease of the gallbladder and of the coronary vessels of the heart may be associated findings.

When hernia is demonstrated in a patient who has attacks of pain in the epigastrium associated with gaseous distention and roentgenoscopic examination discloses stones in the gallbladder, it may be difficult to decide whether one or both of the lesions are responsible for symptoms. If, in addition, electrocardiographic study demonstrates evidence of impairment of coronary circulation, interpretation of the clinical picture is complicated still further.

Lesions of the esophagus that may be mistaken for hernia are cardiospasm, cicatricial stricture, carcinoma and diverticulum above the cardia, or the so-called epiphrenic diverticulum. Ulceration in the lower portion of the esophagus, which has been described as peptic ulcer, results in most instances from congenital shortening of the esophagus with hernia.

TREATMENT

When diaphragmatic hernia of the paraesophageal type is associated with symptoms, operative repair should be recommended if the patient's general condition is satisfactory and discomfort is severe. Even when symptoms are mild, surgical treatment is indicated if the patient is a good operative risk, because there is the likelihood of increase in the size of the hernia, progression of symptoms and a greater risk of mortality as the patient grows older. In no case, however, should operation be undertaken until the esophagus has been investigated completely and found to be free from an obstructive lesion.

When a patient is unsuitable for operation, various procedures may be utilized to promote comfort. If dysphagia is troublesome, passage of a sound, size 60 French, over a previously swallowed silk thread will usually provide complete comfort in swallowing. The relief thus obtained may be permanent, but for some patients occasional passage of a sound is required to maintain normal deglutition. For patients in whom cicatricial stricture has occurred, repeated passage of sounds is necessary to establish and maintain a satisfactory lumen through the esophagus. Avoiding heavy evening meals and sleeping in a semierect position may also diminish nocturnal discomfort.

Results of operative treatment in the management of paraesophageal diaphragmatic hernia are satisfactory, both as to low mortality and as to relief from symptoms. In the treatment of short esophageal hernia, however, placement of the hernial sac below the diaphragm rarely proves beneficial. Because the esophagus is congenitally shortened, recurrence of hernia is frequent; as a matter of fact, dysphagia is usually the most troublesome symptom, and when spasm is the responsible factor the discomfort can almost always be relieved permanently by passage of a size 60 French sound through the esophagus.

When cicatricial stricture is present, repeated dilation of the area of narrowing is required, but relief from dysphagia can always be accomplished. Ulceration at the cardia, with or without bleeding, probably results from retention of a small amount of food or secretion in the lower portion of the esophagus, and when obstruction is relieved by dilation, ulceration usually disappears. In a few instances local treatment of the area of ulceration by application of a solution of silver nitrate may be indicated, but for most patients passage of sounds is the only treatment that is necessary.

SUMMARY

Diaphragmatic hernia occurs with considerable frequency. The most common site of herniation is through the esophageal hiatus. Hernias through this opening are of two main types, the so-called short esophageal and the paraesophageal.

Dysphagia is a frequent and sometimes the most prominent symptom of hernia in this area.

Operative treatment is the most satisfactory procedure in management of the paraesophageal type of hernia. Complete investigation of the esophagus is necessary before surgical treatment, in order to determine accurately the type of herniation and whether or not other lesions are present in the esophagus.

For patients with congenital shortening of the esophagus with hernia, operation is not satisfactory. With hernia of this type, as well as with that of the paraesophageal type, when dysphagia is a prominent symptom and operation is declined or deemed inadvisable, passage of dilating sounds through the esophagus over a previously swallowed silk thread will provide satisfactory results.

ACUTE AND CHRONIC MASTOIDITIS

CLINICAL ANALYSIS OF FIVE HUNDRED AND TWENTY-SIX CONSECUTIVE OPERATIONS

CHARLES EMORY TOWSON, M.D.
PHILADELPHIA

This report consists of an analysis with observations of 526 consecutive operations for acute or chronic mastoiditis which were performed in the services of otology and pediatrics in the same hospital on 466 patients from 1930 to 1939 inclusive. No attempt was made to differentiate between patients treated with sulfonamide compounds and those not so treated, although patients receiving such therapy during the last few years were included. However, the much lower average in the number of operations during the last three years than during the first seven years may be due to the general use of these drugs.

There were 342 mastoidectomies on 288 patients with acute mastoiditis and 179 radical mastoidectomies on 173 patients and 5 modified radical (Heath) mastoidectomies on 5 patients with chronic mastoiditis. Thus 65 per cent of the operations were for acute mastoiditis and 35 per cent for chronic. In addition to those mentioned, 4 patients with mastoiditis were admitted to the hospital and died without operation.

Comparing Kafka's [1] incidence of 65.11 per cent acute and 34.89 per cent chronic mastoiditis in a series of 3,225 cases from Bellevue Hospital in New York with my findings of 65 per cent acute and 35 per cent chronic, one might conclude that in a large city hospital the operations for acute mastoiditis outnumber the radical operations for chronic mastoiditis by about 2 to 1.

INCIDENCE

Lateral Distribution of Mastoiditis.—With the acute type of mastoiditis the incidence on the two sides was equal, each 40 per cent, while with the chronic type the incidence on the left side exceeded that on the right side. Bilateral acute mastoiditis occurred in 18.8 per cent of patients (table 1). Table 2 gives comparative figures for other series.

The term "simple mastoidectomy," while still in common use, is not appropriate, for the reason that many so-called simple mastoidectomies are far from simple. Many are very extensive and complicated. Therefore the term "mastoidectomy for acute mastoiditis," or merely "mastoidectomy," in contradistinction to "radical" or "modified radical" mastoidectomy, is preferable. Lacy [2] said that the term "complete mastoidectomy" should be used instead of "simple mastoidectomy."

Sex.—When the acute and the chronic condition were considered together, the sexes were found to be about evenly divided: Two hundred and thirty-one (49.6 per cent) were males and 235 (50.4 per cent) were females. With the conditions considered separately, the sex incidence was a follows:

Acute: 152 (52.7 per cent) males and 136 (47.3 per cent) females.
Chronic: 79 (44.4 per cent) males and 99 (55.6 per cent) females.

These figures are compared with those of other authors in table 3.

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc., June 1942.

1. Kafka, M. M.: Mortality of Mastoiditis and Cerebral Complications with Review of 3,225 Cases of Mastoiditis, with Complications, Laryngoscope **45**:790 (Oct.) 1935.
2. Lacy, N. E.: Recurring Mastoiditis, Kansas City M. J. **14**:18 (Dec.) 1938.

Age.—The ages of patients operated on for acute mastoiditis ranged from 6 weeks to 70 years, and the ages of those undergoing radical mastoidectomy for chronic mastoiditis varied from 15 months to 67 years (table 4). It will be noted in table 4 that the highest incidence of acute mastoiditis occurred in the age group between 4 and 12 years and the highest incidence of the chronic type in the age group between 19 and 30 years. Table 5 gives comparative figures for other series.

TABLE 1.—*Lateral Distribution of Mastoiditis*

Patients with Acute Mastoiditis	Patients with Chronic Mastoiditis	Total Number of Patients
288 total	178 total	466
117 (40.6%) right	73 (41.1%) right	190 (40.8%) right
117 (40.6%) left	99 (55.6%) left	216 (46.3%) left
54 (18.8%) bilateral	6 (3.3%) bilateral	60 (12.9%) bilateral

TABLE 2.—*Comparison of Lateral Distribution of Acute Mastoiditis as Reported by Other Authors*

Authority	Right, %	Left, %	Bilateral, %
Kreutz and Witter [4]	56.6	34.1	9.3
Kulkin [13]	49.0	41.0	10.0
Kulkin [13] (recurrent)	46.6	51.1	2.3
Tomb [8]	50.0	41.0	9.0
Towson	40.6	40.6	18.8

TABLE 3.—*Comparative Table Showing Sex Distribution of Patients with Acute Mastoiditis*

Authority	Males		Females	
	Number	Per Cent	Number	Per Cent
Kreutz and Witter [4]	160	56.3	131	43.7
Kulkin [13]	128	46.5	147	53.5
Kulkin [13] (recurrent)	25	58.2	18	41.8
Tomb [8]	33	33.0	67	67.0
Towson	152	52.7	136	47.3

TABLE 4.—*Age Distribution of Patients with Mastoiditis*

Age, Yr.	Acute		Chronic		Total	
	No. of Patients	Per Cent	No. of Patients	Per Cent	No. of Patients	Per Cent
0 to 4	87	30.2	3	1.8	90	19.3
4 to 12	93	32.3	32	18.0	125	26.8
12 to 18	29	10.1	27	15.2	56	12.0
18 to 30	27	9.4	66	37.0	93	20.0
30 to 40	18	6.2	26	14.5	44	9.4
40 to 70	34	11.8	24	13.5	58	12.5

HISTORY OF PREVIOUS OTITIS MEDIA AND MASTOIDITIS BEFORE PRESENT ILLNESS

In 65 cases of acute and in 108 cases of chronic mastoiditis there was a history of otitis media previous to the present illness. In 34 cases of acute surgical mastoiditis and in 62 cases in which a radical mastoidectomy was done, a history of mastoidectomy, sometimes multiple, preceded the present operation. In some of the cases the operation had been incompletely done; in others reinfection had occurred, and in others there should have been a radical operation previously.

TIME RELATIONSHIP BETWEEN ONSET OF OTITIS MEDIA AND SURGICAL THERAPY OF MASTOIDITIS

The majority of operations for acute mastoiditis were performed during the second, third or fourth week of the otitis media (table 6). Tomb [3] found by analysis that about the third week was the optimum time for operation as far as complications were concerned.

Occasionally one feeels compelled to operate early, in a few days to a week, either because of an overwhelming infection or—as is more often the case—

TABLE 5.—*Comparative Table Showing Age Distribution for Mastoiditis in Several Series of Cases*

Authority *	Type of Mastoiditis	Age Range
Kreutz and Witter [4]	Acute	3 mo. to 68 yr.
Tomb [3]	Acute	5 mo. to 58 yr.
Persky [24]	Chronic	23 mo. to 56 yr.
Towson	Acute	6 wk. to 70 yr.
Towson	Chronic	15 mo. to 67 yr.

* Of Kulkin's [18] patients with acute mastoiditis, 44.7 per cent were 5 years old or less and 79.3 per cent 10 years old or less. Of his patients with acute or recurrent mastoiditis, 36 (1.13 per cent) were up to 1 year of age, and 1 was over 50 years old. Of Tomb's [3] patients with acute mastoiditis, 24 (24 per cent) were under 5 years of age and 29 (29 per cent) between 5 and 10 years of age.

TABLE 6.—*Time Relationship Between Onset of Otitis Media and Surgical Therapy of Mastoiditis*

Operation for Acute Mastoiditis				Radical Mastoidectomy			
Time	No. of Cases	Time	No. of Cases	Time	No. of Cases	Time	No. of Cases
1 day	2	3 mo.	10	1 day	1	4 yr.	6
2 days	1	4 mo.	3	3 days	1	5 yr.	4
3 days	4	6 mo.	2	4 days	1	6 yr.	5
4 days	6	9 mo.	1	5 days	1	7 yr.	3
5 days	7	10 mo.	1	1 wk.	3	8 yr.	6
6 days	1	1 yr.	1	2 wk.	5	9 yr.	5
7 days	26	1½ yr.	2	3 wk.	5	10 yr.	12
8 days	1	2 yr.	3	1 mo.	5	11 yr.	3
9 days	2	3 yr.	1	2 mo.	4	12 yr.	4
10 days	13	4 yr.	1	3 mo.	11	13 yr.	5
11 days	2	5 yr.	3	4 mo.	4	14 yr.	4
12 days	8	6 yr.	2	5 mo.	2	15 yr.	6
2 wk.	46	8 yr.	1	6 mo.	2	16 yr.	4
3 wk.	48	9 yr.	1	7 mo.	1	18 yr.	1
4 wk.	36	12 yr.	1	8 mo.	3	19 yr.	2
5 wk.	9	14 yr.	1	9 mo.	1	20 yr.	3
6 wk.	5	24 yr.	1	10 mo.	2	21 yr.	1
7 wk.	8	45 yr.	1	12 mo.	6	23 yr.	3
2 mo.	15	No otitis media	12	14 mo.	1	25 yr.	3
				18 mo.	5	30 yr.	2
				24 mo.	6	50 yr.	2
				30 mo.	1	? yr.	8
				3 yr.	10	Unknown	4

because the actual beginning of the otitis media was two or three weeks previous to the stated onset, and was so mild that it was overlooked by the family, and the actual mastoid infection continued without signs or symptoms. The last-mentioned fact accounts, in most cases, for the so-called primary mastoiditis, which apparently develops without otitis media. Patients with this condition, when first seen by the physician, present at times marked swelling and fluctuation over the mastoid, while the ear drum appears normal.

In spite of the optimum time for surgical therapy, many patients, even with a goodly amount of destruction within the mastoid, will go along apparently in

3. Tomb, E. H.: Simple Mastoidectomy: A Critical Analysis of One Hundred Consecutive Cases, Arch. Otolaryng. **31**:478 (March) 1940.

good health for two to three months before operation and will have an uneventful and easy convalescence.

The time element for the patients with radical operations varied from one day to fifty years or more (table 6). Those operated on in less than a week after onset had acute attacks superimposed on chronic mastoiditis.

SEASONAL VARIATION

In table 7 it will be seen that the greater number of operations for acute mastoiditis were done during the period from midwinter through late spring and early summer. Most of the patients with chronic mastoiditis were operated

TABLE 7.—*Seasonal Variation in Incidence of Surgical Mastoiditis*

Month	Acute Mastoiditis		Chronic Mastoiditis		Total with Mastoiditis	
	Number	Per Cent	Number	Per Cent	Number	Per Cent
January	24	8.3	9	5.1	33	7.1
February	33	11.5	11	6.2	44	9.4
March	41	14.2	9	5.1	50	10.7
April	45	15.6	16	9.0	61	13.1
May	31	10.8	15	8.4	46	9.9
June	30	10.4	18	10.1	48	10.3
July	18	6.3	20	11.2	38	8.2
August	14	4.9	19	10.7	33	7.1
September	6	2.1	21	11.8	27	5.8
October	13	4.5	17	9.5	30	6.4
November	13	4.5	11	6.2	24	5.2
December	20	6.9	12	6.7	32	6.8

TABLE 8.—*Etiologic Factors in the Original Otitis Media*

Cause	Number of Patients	
	Acute Mastoiditis	Chronic Mastoiditis
Head cold	107	12
Bronchopneumonia	16	..
Measles	12	15
Grip	10	1
Bronchitis	7	..
Swimming	6	7
Teething	5	1
Lobar pneumonia	3	1
Tonsillitis	3	..
Scarlet fever	3	15
Other factors	18	14
Undetermined	98	112
Total number	288	178

on during the summer months. This was probably due to the fact that many of the latter operations, the need not being urgent, were done at the convenience of the surgeon.

ETIOLOGY

It was difficult many times, especially in the case of a very chronic mastoiditis, to obtain an accurate history of the exact cause of the original infection, either from the patient or from the parents. Hence table 8 is not as accurate as it might be. Evidently, in most cases, infections of the upper and lower respiratory tract, measles and scarlet fever predominated as the primary infection.

Kreutz and Witter,[4] in a study of 300 cases of acute mastoiditis, stated that in 42.5 per cent there was "frank clinical evidence of paranasal sinus disease."

4. Kreutz, G. C., and Witter, G. L.: Observations in Three Hundred Cases of Acute Mastoiditis, Ann. Otol., Rhin. & Laryng. **46**:1060 (Dec.) 1937.

Campbell [5] found associated sinus disease in 150 cases of acute otitis media in infants and children. Fowler [6] stated that sinus disease was important as a cause of chronic and recurrent suppurative otitis media and reported such infection in 86 per cent of cases of otitis media.

Of 564 cases of suppurative otitis media in children under 5 years of age, a cause of onset other than acute corzya was assigned and recorded by Scott and Lumsden [7] in 46. Tomb,[8] in a study of 100 cases of acute mastoiditis, gave the preceding illness as follows: in 51 per cent, infections of the upper respiratory tract; in 5 per cent, acute tonsillitis, and in 3 per cent, sinusitis. Kopetzky [8] agreed with Hadjopoulos and Bell,[9] who expressed the opinion that the source of infection of the mastoid cells lies in the blood and lymph channels of the adjacent mucosa. Bolotow [10] found that in 26 cases in which a radical mastoidectomy was performed the infection dated back to: scarlet fever, in 9 cases; measles, in 6 cases; scarlet fever and measles, in 1 case; diphtheria, in 1 case; head colds, in 6 cases, and tonsillitis, in 3 cases.

Williams,[11] in a study of 14,733 cases of scarlet fever and 1,954 cases of measles, concluded that sinusitis is a large factor in the production of otitis media. Veeder [12] stated:

> It is difficult for me to associate sinusitis and otitis media as cause and effect . . . I think that the two conditions are due to the same cause . . . and the sinus becomes involved at the same time.

BACTERIOLOGY

About twenty-two different organisms were isolated in the whole series. In 29 cases there was no growth, and in 168 cases no record was available. Streptococcus haemolyticus was by far the most frequent. Streptococcus in combination with one or more other organisms ranked next highest, with the pneumococcus third (table 9).

Kreutz and Witter,[4] Kulkin,[13] Ebbs [14] and Tomb [3] all reported the hemolytic streptococcus to be the most common organism.

METHOD OF DRAINAGE OF THE MIDDLE EAR

When the methods of drainage of the middle ear during the attack of otitis media preceding mastoiditis (table 10) were considered, the incidence of spontaneous rupture of the ear drum was found to exceed by far that of any other method, the number of cases of acute mastoiditis in which it occurred being 118. The method with the next highest incidence was single myringotomy, in 69 cases. Likewise, in the cases of chronic mastoiditis spontaneous rupture of the ear

---

5. Campbell, E. H.: Association of Acute Sinusitis and Acute Otitis Media in Infants and Children, Arch. Otolaryng. **16**:829 (Dec.) 1932.

6. Fowler, E. P.: Incidence of Nasal Sinusitis with Diseases of the Ear, Arch. Otolaryng. **9**:159 (Feb.) 1929.

7. Scott, C. E., and Lumsden, R. B., in Discussion on Otitis Media in Early Childhood (Under Five Years), Proc. Roy. Soc. Med. **30**:1293 (Aug.) 1937.

8. Kopetzky, S. J.: Purulent Otitis Media, Sinus Thrombosis and Suppuration of the Petrous Pyramid: Acute and Chronic Cases, Arch. Otolaryng. **28**:626 (Oct.) 1938.

9. Hadjopoulos, L. G., and Bell, J. W., cited by Kopetzky.[8]

10. Bolotow, N. A.: Radical Mastoidectomy: Its Use in Treatment of Dangerous Types of Suppuration of the Temporal Bone, Arch. Otolaryng. **29**:269 (Feb.) 1939.

11. Williams, H. J.: Diagnosis and Treatment of Diseases of the Ear in Children, J. A. M. A. **113**:990 (Sept. 9) 1939.

12. Veeder, B. S., in discussion on Williams.[11]

13. Kulkin, S.: Acute Mastoiditis from the Surgical Point of View, Arch. Otolaryng. **29**: 306 (Feb.) 1939.

14. Ebbs, J. H., in Discussion of Otitis Media in Early Childhood (Under Five Years), Proc. Roy. Soc. Med. **30**:1293 (Aug.) 1937.

drum headed the list. If this means anything at all, one might conclude that proper myringotomy is better prophylaxis than spontaneous rupture.

There has always been some argument as to whether incision of the drum membrane has any notable advantage over spontaneous rupture in preventing mastoiditis. However, most authorities are agreed that myringotomy is better. Griffin [15] expressed preference for early incision of the drum membrane as a preventive measure. Kreutz and Witter [4] stated:

> Paracentesis also was advantageous in that (1) it afforded earlier relief of pain and allowed rest; (2) it permitted the collection of uncontaminated cultures from the middle ear cavity; (3) it tended to lessen complications, for [in their series of 300 acute mastoidectomies] only eleven cases developed complications, eight recovering and three dying, contrasted with thirty-seven complications, thirteen of them fatal, when the drums were allowed to rupture.

Tomb [8] also stated the belief that myringotomy is advisable.

TABLE 9.—*Types of Organisms Isolated*

Organism	Acute Mastoiditis	Chronic Mastoiditis
Str. haemolyticus	124	9
Streptococcus of various types with one or more other organisms	26	9
Pneumococcus	17	3
Staphylococcus albus	9	5
Staphylococcus aureus	4	5
Streptococcus viridans	3	3
Bacillus pyocyaneus	3	2
Diphtheroid bacillus	2	0
Bacillus proteus	1	8
Friedländer's bacillus	2	1
Bacillus coli communis	1	0
All other combinations	10	13
No growth	15	14
No record	62	106

TABLE 10.—*Type of Opening of the Drum Membrane in the Attack of Otitis Media Preceding Mastoiditis*

Type of Opening	Acute Mastoiditis	Chronic Mastoiditis
Spontaneous rupture	118	130
Single myringotomy	60	10
Multiple myringotomies (two or more)	35	3
Spontaneous rupture and single myringotomy	31	5
Spontaneous rupture and multiple myringotomies	10	3
No opening in drum membrane	15	..
Unknown	10	27

Williams,[11] in two large series of cases of otitis media complicating scarlet fever and measles, reported the occurrence of surgical mastoiditis in 6.6 and 4.4 per cent respectively of cases in which primary myringotomy was done, in 7.7 and 2.77 per cent of cases in which myringotomy was employed after spontaneous rupture and in 9.3 and 11 per cent of cases in which drainage was by spontaneous rupture alone.

In a previous study [16] I concluded: "Early surgical drainage of the middle ear cavity by myringotomy in the presence of a bulging drum head is good practice."

Stuart,[17] in a study of 754 patients with acute suppurative otitis media, wrote that: "31 per cent of the patients requiring mastoidectomy had paracentesis of the tympanic membrane during the first week of disease, whereas 68 per cent of the patients who did not require mastoidectomy had similar treatment."

15. Griffin, E. A., cited by Kafka.[1]

16. Towson, C. E.: Aural Complications of Pneumonia in Children, J. Pediat. **11**:77 (July) 1937.

17. Stuart, E. A.: Acute Suppurative Otitis Media: A Review of Cases Encountered During a Five Year Period, Arch. Otolaryng. **33**:734 (June) 1941.

LEUKOCYTOSIS

In table 11 it will be seen that most of the patients with acute mastoiditis had a leukocyte count between normal and 15,000 per cubic millimeter. The higher counts occurred in very ill patients or in those with complications. The majority of those with chronic mastoiditis had a white blood cell count below 12,000, and many of them had a normal count, which of course is to be expected.

TEMPERATURE

Most of the patients with acute mastoiditis had a temperature range below 104 F. (table 12). The figures in the table indicate the high points reached either before or after operation. The average temperature with acute mastoiditis ranged between 100 and 103 F. If in any case it was above 103 F. a complication of some sort had to be considered.

TABLE 11.—*Variation in White Blood Cell Count of Patients with Mastoiditis*

White Blood Cell Count	Acute Mastoiditis	Chronic Mastoiditis
Below 5,000	2	10
5,000 to 10,000	62	90
10,000 to 12,000	31	21
12,000 to 15,000	66	16
15,000 to 20,000	66	7
20,000 to 25,000	19	2
25,000 and over	10	1
No record	32	31

TABLE 12.—*Temperature Range for Patients with Mastoiditis*

Temperature	Acute Mastoiditis	Chronic Mastoiditis
99 to 100 F	21	35
100 to 101 F	53	74
101 to 102 F	46	26
102 to 103 F	40	16
103 to 104 F	46	10
104 to 105 F	37	8
105 to 106 F	24	5
106 to 107 F	18	4
107 to 108 F	2	..
108 to 109 F	1	..

The temperature of patients with chronic mastoiditis without complications was much lower, many of them having only 99 F. both before and after operation.

ROENTGENOGRAPHIC OBSERVATIONS

While roentgenograms are not infallible, and most workers agree that they should not be the final word in the diagnosis, they are valuable and should be taken in every case. In rare cases the roentgenogram will be the deciding factor calling for surgical intervention when all other evidence is against it. Certain valuable information will be given by roentgenograms, regarding (1) type of cell structure and the size and extent of the mastoid; (2) the kind and amount of pathologic change, whether the trabeculae are thick, thin, hazy or absent and whether there is cavitation (a competent roentgenologist can tell many times whether there is pus or cholesteatoma in a cavity); (3) visibility of the sinus plate, with its location, size and possible erosions, and (4) the anatomic structure and state of the petrous pyramid.

In this series there were only 18 cases in which the roentgenographic observations disagreed with the observations at operation. The roentgenographic diagnosis

was normal in 13 cases in which the disease was found at operation. In 2 cases the mastoid was apparently normal when the roentgenogram showed disease. Disease was found at operation in 3 cases of chronic mastoiditis in which the roentgenologic report was normal (table 13).

Bolotow,[10] with regard to 26 cases of chronic mastoiditis in which operation was performed, stated: "In nearly all cases the operative findings tallied with the results of the preliminary roentgen studies." Ashley [18] stated: "Roentgen rays are of little value in diagnosis [in chronic suppurative otitis media], because of the sclerosis so frequently present." Kulkin,[13] in a study of 275 cases of acute mastoiditis (first attack), stated: "Roentgen reports in every case of acute mastoiditis indicated clouding of the mastoid cells, cellular destruction and exudate."

GENERAL PROCEDURE IN RADICAL MASTOIDECTOMIES

In a small number of radical mastoidectomies the Smythe periosteal flap was used. As a rule, in cases in which this procedure was employed, dermatization took place in a relatively shorter period. However, the Panse flap, or modification of it, was used in the great majority of cases. Every effort was made to remove all perilabyrinthine cells, not only in the radical mastoidectomy but in the operation for the acute type when indicated, and I believe this procedure in part

TABLE 13.—*Roentgenographic Observations on Patients with Mastoiditis*

Roentgenographic Observations	Acute Mastoiditis	Chronic Mastoiditis
Cloudiness	130	17
Destruction	27	20
Sclerosis	24	89
Normal	13	3
Motion on film	5	..
No record	89	49

accounted for the relatively few cases of petrositis in this series. All mastoid cells wherever situated were removed in cases both of the acute and of the chronic type of mastoiditis. The middle ear was thoroughly cleaned out, but the eustachian tube was not curetted in most cases. In most of the radical operations the opening was closed up tight posteriorly, with the periosteum sutured separately from the skin. The closing of the periosteum afforded a stronger wall and gave better healing. The making of a large, rather than a small, meatal opening allowed better and easier postoperative care. There have been times when I wished a larger opening had been made.

In nearly all the radical operations the cavity was packed with iodoform gauze drain. This was removed and changed on the fourth day as a rule. After a few dressings plain sterile gauze, or gauze saturated with acriflavine, was used. The wound was dressed every day, or every other day, for a while, depending on how much discharge was present. At times petrolatum gauze was used, and for a foul discharge gauze saturated with potassium permanganate. Dusting powder was employed at times to dry the cavity. Excessive granulations were kept down by applications of silver nitrate or chromic acid, or even by curettement. Roentgen treatment was used in some cases to inhibit granulations, with good results. Some patients had to be anesthetized and the granulations cleaned out again, and occasionally several times. The frequency of dressings and the length of time

18. Ashley, R. E.: Surgical and Nonsurgical Care of the Chronically Discharging Middle Ear, Arch. Otolaryng. **33**:993 (June) 1941.

the wound was packed depended on circumstance. I believe physicans who follow the patients personally will agree that no dogmatic rule can be followed. Each one must be treated according to the judgment of the operator. Some wounds will heal and dermatize quickly if the cavity is packed only once or twice after operation, and others will not but must be packed for a longer time. Sometimes excessive packing seemed to stimulate granulations; yet some wounds filled in too much, and pockets of pus formed when they were not packed. One requires a great deal of experience to know what procedure to follow in all cases.

EXPOSURE OF LATERAL SINUS AND DURA

The total number of exposures of the lateral sinus was 222 and the total number of exposures of the dura 240. In some cases both structures were exposed, and in the cases of bilateral mastoiditis there were various combinations. It is unfortunate that there was not sufficient information in the histories to indicate in what percentage of cases the exposure was due to necrosis, in what per-

TABLE 14.—*Exposure of Lateral Sinus and Dura*

	Acute Mastoiditis		Chronic Mastoiditis	
	Unilateral	Bilateral	Unilateral	Bilateral
Sinus	31	2, both sides 7, one side only	18	
Dura	33	5, both sides 5, one side only	26	1, both sides 1, one side only
Dura and sinus	61	6, both sides 4, one side only 1, dural exposure on one side; sinus exposure on opposite side 1, dural and sinus exposure on one side, dural exposure only on opposite side	77	2, both sides 2, one side only
None	110	22	51	

centage the exposure was intentional for investigation and in how many it was accidental. It can safely be said, however, that in over 50 per cent of cases the exposure was accidental or for investigation of normal membranes. In none of these cases did any complication develop as a result of the exposure. In all cases in which a small exposure was made accidentally, the area was enlarged to a goodly size. I believe such a procedure is necessary to prevent the spreading of infection through a very small opening in the bone, through which there will not be proper drainage. I believe the sinus and dura can be exposed without danger provided the exposure is made large enough.

Kriegsman,[19] however, did not agree. Kafka said that Kriegsman's "Conclusion is that in uncovering the sigmoid sinus, it is apt to invite a hazard more frequently than is generally accepted. . . . He thinks that exposure of the vein, without well founded suspicion for disease should be discarded."

Information is lacking in the records, but I recall from memory a few cases of exposure of the facial nerve by necrosis, and in at least 1 of these cases there was no paralysis or weakness of the face either before or after operation.

19. Kriegsman, G., cited by Kafka.[1]

COMPLICATIONS

In 55, or in 19 per cent, of the cases of acute mastoiditis, there were intracranial complications, and in 69, or 23.9 per cent, there were other complicating diseases of nonotitic origin. In 36, or in 20.2 per cent, of the cases of chronic mastoiditis there were intracranial complications, and in 16, or 8.9 per cent, there were other complicating diseases of nonotitic origin (table 15).

Kafka [1] found cerebral complications in 6 per cent of 2,100 cases of acute mastoiditis and in 7.37 per cent of 1,125 cases of chronic mastoiditis; Kreutz and Witter,[4] in 16 per cent of 300 cases of acute mastoiditis, and Kulkin,[13] in 3.4 per cent of 318 cases of acute or recurrent mastoiditis. Of 100 consecutive cases of surgical mastoiditis reported by Johnson,[20] in 4 per cent there were complications of otitic origin before operation, and in 36 per cent complications developed after operation.

CHOLESTEATOMA

In this series of cases a radical mastoidectomy was performed on a child only 15 months of age because of the presence of cholesteatoma. It is unusual to find cholesteatoma in a child so young and, further, to perform a radical mastoidectomy

TABLE 15.—*Intracranial Complications* in Cases of Mastoiditis*

Complications	Acute Mastoiditis	Chronic Mastoiditis
Perisinus abscess	34	13
Sinus phlebitis	3	..
Extradural abscess	5	5
Extracerebellar abscess	3	5
Brain abscess	3	4
Cerebellar abscess	1	3
Sinus thrombosis	5	4
Meningitis	8	10
Facial paralysis	2	4
Petrositis	1	5
Labyrinthitis	1	6
Fistulous labyrinth	..	1
Lateral sinus obliterated (of long duration without symptoms)	..	2

* Complications are listed in 66 cases of acute mastoiditis and in 62 cases of the chronic form. In several cases there was more than one complication.

on a patient of this age. Without discussing the primary and secondary types of cholesteatoma, as described by Tumarkin,[21] I can say that in this case the tumor was of the secondary type. The aural infection was of thirteen and one-half months' duration and was therefore chronic.

I have found by trial and error that radical mastoidectomy must be done in cases of cholesteatoma. If, as Lange [22] stated, and in accordance with all other observers (Bolotow [10]), including Kopetzky,[8] cholesteatoma is confined to the epitympanum, a modified radical operation may be sufficient. But if the tumor occupies the rest of the tympanum, nothing but a complete radical operation will suffice. The authors just mentioned agreed that cholesteatoma is always a threat to life. Young [23] "has come to the conclusion that unless certain exceptional circumstances exist, the presence of cholesteatoma demands as radical an operation as possible."

20. Johnson, H. P.: Study of One Hundred Consecutive Mastoidectomies, Maine General Hospital: Temperature After Mastoidectomy, Arch. Otolaryng. **19**:660 (June) 1934.

21. Tumarkin, A.: A Contribution to the Study of Middle Ear Suppuration with Reference to the Pathology and Treatment of Cholesteatoma, J. Laryng. & Otol. **53**:685 (Nov.); 737 (Dec.) 1938.

22. Lange, cited by Kopetzky.[8]

23. Young, G., in discussion on Hastings.[26]

Cholesteatoma was found in 5 patients who had so-called simple mastoidectomies, the end results in which show the necessity for radical surgical intervention: One patient had a radical operation three years later; 1, six years later, and 1, two years later. One patient reported that he had had no further operation on the mastoid but the discharge continues. One patient reported that the mastoid healed in three months, with no further trouble.

In 85 of the cases in which radical mastoidectomy was performed cholesteatoma was encountered, a number which is nearly 50 per cent of the cases of chronic mastoiditis. Persky [24] reported the observation of cholesteatoma in 21.2 per cent of 151 cases in which radical mastoidectomy was performed. Ruedi [25] stated that of 763 cases of chronic suppuration of the middle ear encountered at the University Clinic at Zurich, Switzerland, cholesteatoma was found in every one. In one third of these cases the disease followed scarlet fever, measles or diphtheria. Tumarkin [21] stated that in one third of all cases of otitis media cholesteatoma develops.

TABLE 16.—*Analysis of Mortality in Cases of Acute Surgical Mastoiditis*

Age	Diagnosis	Cause of Death
13 mo.	Bilateral mastoiditis; microcephalus; atrophy of the brain.	Atrophy of the brain
9 yr.	Bilateral mastoiditis; meningitis	Meningitis
8 mo.	Bilateral mastoiditis; bronchopneumonia	Bronchopneumonia
8 yr.	Mastoiditis (right); cerebellar abscess; acute meningitis	Acute meningitis
9 mo.	Mastoiditis (right); cellulitis of right arm (caused by vaccination); streptococcic infection of blood stream	Septicemia
9 yr.	Mastoiditis (left); pulmonary edema	Pulmonary edema
3 mo.	Mastoiditis (left); bronchopneumonia	Bronchopneumonia
10 mo.	Mastoiditis (left); bronchopneumonia	Bronchopneumonia
7 mo.	Mastoiditis (left); miliary tuberculosis; acute generalized tuberculous meningitis	Tuberculous meningitis
9½ mo.	Mastoiditis (left); bronchopneumonia; sinus thrombosis on left	No autopsy; death probably due to sinus thrombosis
47 yr.	Mastoiditis (left); malignant process in left temporal bone; localized meningitis	Meningitis
10 yr.	Mastoiditis (left); septic sinus thrombosis; septicemia; phlebitis (left leg); bronchial foci: meningitis	Meningitis
31 yr.	Mastoiditis (left); meningitis	Meningitis
16 yr.	Mastoiditis (left); acute meningitis	Meningitis
70 yr.	Bilateral mastoiditis; erysipelas	Erysipelas (and renal failure)
Child	Bilateral mastoiditis; pneumonia	Pneumonia
43 yr.	Mastoiditis (right); meningitis	Meningitis
18 yr.	Mastoiditis (left); acute suppurative meningitis	Meningitis
13 yr.	Mastoiditis (left); acute purulent meningitis; fascial abscess in left subtemporal region following zygomatic mastoiditis	Meningitis

MORTALITY

The deaths of 27 of the 466 patients operated on constituted a mortality rate of 5.8 per cent. Of the patients who were operated on for acute mastoiditis, 19, or 6.6 per cent, died. Of these, 5 had the disease on both sides, 3 on the right side and 11 on the left side. Of those undergoing radical mastoidectomy, 8, or 4.5 per cent, died; 1 had the disease on both sides, 4 on the right side and 3 on the left side (tables 16, 17 and 18). From tables 16 and 17 it is evident that meningitis was the predominating cause of death.

HOSPITALIZATION

The average patient with acute mastoiditis, without complications and with good resistance, spent from ten days to three weeks in the hospital. Many were in

24. Persky, A. H.: The Problem of the Radical Mastoid: A Critical Analysis of One Hundred and Fifty-One Cases, Pennsylvania M. J. **42**:231 (Dec.) 1938.

25. Ruedi, L.: Zur Klinik und Therapie des Mittelohrcholesteatoms, Schweiz. med. Wchnschr. **64**:411 (May 12) 1934.

much longer, a few several months. In general, the chief reasons for a long stay in the hospital were: (1) the patient was severely ill; (2) the patient's home was too far away for him to receive proper attention, and (3) home conditions were inadequate for the best welfare of the patient.

The majority of the patients with radical mastoidectomies stayed in the hospital two to six weeks. Many lived out of the city, which necessitated longer hospitalization for proper postoperative care, when otherwise they could have been out of the hospital within a week. The reasons for long hospitalization were the same as those for the cases of acute mastoiditis.

TABLE 17.—*Analysis of Mortality in Cases of Radical Mastoidectomy for Chronic Mastoiditis*

Age	Diagnosis	Cause of Death
44 yr.	Bilateral mastoiditis; bilateral labyrinthitis (suppurative on left side); chronic suppurative extracerebral and cerebellar and perisinus abscess on right side; pachymeningitis, left cervical adenitis (suppurative)	Meningitis
26 yr.	Mastoiditis (right); extradural abscess	Cerebellar abscess
33 yr.	Mastoiditis (right); basilar meningitis	Meningitis
35 yr.	Mastoiditis (right); postoperative meningitis; extradural abscess	Meningitis
46 yr.	Mastoiditis (right); meningitis	Meningitis
44 yr.	Mastoiditis (left); acute suppurative meningitis; osteomyelitis of skull; degeneration of kidney	Meningitis
10 yr.	Mastoiditis (left); septic sinus thrombosis, suppurative basal meningitis; cerebellar abscess; bronchopneumonia; pulmonary abscess; cholesteatoma	Pneumonia and meningitis
3 yr.	Mastoiditis (left); osteomyelitis of skull; cervical abscess	Cardiac failure during tonsillectomy

TABLE 18.—*Mortality Rates for Operations for Acute and Chronic Mastoiditis Reported by Other Authors*

Author	Mastoiditis	Mortality Rate, per Cent
Kafka [1]	2,100; acute	4.28
Kafka [1]	1,125; chronic	5.80
Dixon *	186	6.90
Dixon	12	16.60
Dixon	60	10.00
Dixon	77	6.40
Ruedi [25]	342; chronic	1.18
Kreutz and Witter [4]	300; acute	6.30
Kulkin [13]	318; acute	1.20
Johnson [20]	100	2.00
Tomb [3]	100; acute	1.00
Roberts †	174; acute	6.30

* Dixon, J. O., cited by Kafka.[1]

† Roberts, E. R.: A Consideration of Acute Purulent Otitis Media. J. Connecticut M. Soc. 1:368 (Aug.) 1937.

ANSWERS TO QUESTIONNAIRE

Four hundred and twenty-three follow-up questionnaires were sent out, 224 of which were returned because the postoffice department failed to locate the addressee. To the remaining 199 there were 134 responses, which is a fair percentage. Table 20 is presented to show how the patient feels about the end results of the operation.

HEALING TIME OF THE MASTOID AND PERCENTAGE OF DRY EARS

With the omission of 19 patients who died and 64 patients for whom I have no data, the records showed that all but 4, or 98 per cent, of those who were operated on for acute mastoiditis had dry ears. With the omission of 8 who died and 47 for whom I have no record, all but 12, or 90 per cent, of the patients who had radical operations had dry cavities. These figures seem high, probably because for some of the patients who were not followed up (approximately 25

per cent), the outcome, if it were known, would be found to be unfavorable, thus reducing the percentage of good results.

Hastings[26] reported on the percentage of dry ears obtained from several studies. His figures are presented in table 21, together with those from three other sources.[27]

The healing time for patients operated on for acute mastoiditis varied from two weeks to one year; for those with radical mastoidectomies it varied from two months to eight years (table 22).

TABLE 19.—*Period of Hospitalization After Mastoidectomy*

Time of Hospitalization	Operation for Acute Mastoiditis	Radical Mastoidectomy
1 day	2 (2 died)	..
3 days	1 (1 died)	..
5 days	3	..
6 days	..	2
7 days	13 (2 died)	..
8 days	6	6
9 days	2	4
10 days	10	9 (2 died)
11 days	11	7
12 days	11 (2 died)	8
13 days	2	..
14 days	40 (3 died)	28
15 days	29	19 (2 died)
3 weeks	38 (2 died)	27 (1 died)
4 weeks	30 (1 died)	16 (1 died)
5 weeks	20 (2 died)	11
6 weeks	10	10 (1 died)
7 weeks	12 (2 died)	7
8 weeks	18 (2 died)	4
9 weeks	3	4
10 weeks	6	3
11 weeks	2	1
12 weeks	3	2
3 months	10	3
4 months	4	3
5 months	2	2
8 months	..	2 (1 died)

TABLE 20.—*Answers to Questionnaire*

	Operation for Acute Mastoiditis	Radical Mastoidectomy
Total replies	64	70
Total cures	58	51
Aural discharge continues	3	13
Postoperative hearing better	12 (18%)	24 (34%)
Postoperative hearing same	42 (30 normal, 65%)	17 (24%)
Hearing worse	10 (17%)	29 (41%)
Postoperative tinnitus	27	36
Health improved	48	52
Health same (good)	12	6
Health not improved	4	12

RESIDUAL HEARING

Unfortunately, complete records of the hearing were not kept for all patients. Figures in table 23 were compiled from the hospital records, audiograms and answers to the questionnaire. Of the patients with radical mastoidectomies, 63.8 per cent heard as well or better after operation.

Table 25, compiled from audiograms and statements made by patients themselves, gives information in regard to the hearing before and after operation. This

26. Hastings, S., in Discussion on the Operative Treatment of Chronic Mastoid Disease, Proc. Roy. Soc. Med. **31**:331 (Dec.) 1937.

27. (*a*) Cited by Hastings.[26] (*b*) McKenzie.[28] Bolotow.[10] Maxwell and Richter.[29]

information was obtained from 66 of the patients having operations for acute mastoiditis and from 94 of those having radical mastoidectomies.

Table 26 gives information about 30 patients with radical mastoidectomies, who, in response to the questionnaire, gave their estimates of the hearing before and after operation and who returned recently for an audiogram. It is unfortunate that no records could be found of preoperative audiograms. The table also shows the duration of otorrhea and the healing time in each case.

TABLE 21.—*Percentage of Dry Ears Reported by Other Authors*

Operation	Reporter	Percentage of Dry Ears
Simple mastoidectomy	Hastings [26] (Middlesex cases)	75
Conservative mastoidectomy	Howarth and Bateman [27a]	43
Atticoantrotomy	Carter [27a]	65
Atticoantrotomy	Birrell [27a]	82
Atticoantrotomy	Jessen [27a]	58
Atticoantrotomy	Hastings [26] (Middlesex cases)	25
Radical mastoidectomy	Fraser [27a]	42
Radical mastoidectomy	Jessen [27a]	32
Radical mastoidectomy	White [27a]	57
Radical mastoidectomy	Hastings [26] (Middlesex cases)	46
Atticoantrotomy	McKenzie [28]	35
Radical mastoidectomy	Bolotow [10]	84
Radical mastoidectomy	Maxwell and Richter [29]	84

TABLE 22.—*Healing Time After Mastoidectomy*

Operation for Acute Mastoiditis	No. of Patients	Radical Mastoidectomy	No. of Patients
2 weeks	2	2 months	11
3 weeks	8	3 months	12
4 weeks	48	4 months	9
5 weeks	5	5 months	12
6 weeks	32	6 months	11
7 weeks	6	7 months	3
8 weeks	36	8 months	1
3 months	25	9 months	4
4 months	24	10 months	1
5 months	9	12 months	21
6 months	3	15 months	1
10 months	1	18 months	4
1 year	2	2 years	16
Continuous discharge	4	2½ years	1
Death	19	3 years	2
Result unknown	64	5 years	1
		8 years	1
		Continuous discharge	12
		Death	8
		Result unknown	47

TABLE 23.—*Postoperative Hearing in Cases of Mastoiditis*

	Hearing Better	Hearing Unchanged	Hearing Worse
Acute mastoiditis	21.2%	66.7%	12.1%
Chronic mastoiditis	29.8%	34.0%	36.2%

Table 27 shows preoperative and postoperative loss of hearing in a few patients as shown with the audiogram. In addition, the duration of otorrhea before operation and the healing time of the mastoid are shown.

From an analysis of tables 26 and 27, one may conclude that the healing time of the mastoid evidently had no bearing on the resultant hearing. The relationship between the end result in hearing and the duration of otorrhea was such that one might conclude that the longer the discharge the worse the residual hearing. Bolotow [10] stated the following belief:

> The improved hearing . . . undoubtedly resulted from good receptive power in the cochlea and diligent postoperative care to prevent heavy granulations from accumulating in the region

of the posterior tympanic wall. The development of a thin epidermal layer over the promontory permits better reception of sound.

If a patient with chronic otorrhea is given conservative and expectant treatment for several years, without improvement, and the hearing is getting worse, I believe the progressing deafness in many cases can be checked by an operation. Young [23] also claimed that further deterioration in the hearing in such a patient can be prevented by surgical intervention.

McKenzie,[28] in a series of 50 cases of atticoantrotomy, reported good hearing in 34 per cent, fair hearing in 30 per cent, poor hearing in 29 per cent, improved hearing in 57 per cent, no change in 28 per cent and poorer hearing in 15 per cent.

Maxwell and Richter,[29] reviewing the results of Keen, stated:

> Keen concluded that the . . . best postoperative hearing was obtained for patients with a thin, reddish, dry lining of the radical mastoidectomy cavity. The poorest hearing was found

TABLE 24.—*Postoperative Hearing Reported by Hastings* [26] *and Other Authors* [27a]

Operation	Author	Hearing Better, %	Hearing Unchanged, %	Hearing Worse, %
Simple mastoidectomy	Hastings	50	38	12
Atticoantrotomy	Carter	43	35	22
Atticoantrotomy	Jessen	40	26	26
Atticoantrotomy	Hastings	12	50	38
Radical mastoidectomy (grafted)	Fraser	72	14	14
Radical mastoidectomy (nongrafted)	Fraser	64	11	25
Radical mastoidectomy	Pressman	35	18	47
Radical mastoidectomy	Hastings	31	38	31

TABLE 25.—*Condition of Hearing Before and After Operation*

Hearing Before Operation	Hearing After Operation	Acute Mastoiditis	Radical Mastoiditis
Normal	Normal	53.0%	2.1%
Normal	Impaired	7.0%	17.1%
Impaired	Normal	16.7%	1.1%
Impaired	Improved but not normal	4.5%	28.7%
Impaired	Same	13.7%	31.9%
Imparied	Worse	4.5%	19.1%

in those with an open eustachian tube and a moist thick lining of mucous membrane in the middle ear.

White [30] concluded that the best hearing results were obtained for persons with chronic suppurative otitis media of five to fifteen years' standing. In 37 per cent of the series studied hearing was improved, in 14 per cent it was unchanged and in 49 per cent it was made worse.

Maxwell and Richter [29] stated:

> Fowler . . . maintained that the average radical mastoidectomy does not benefit the hearing. . . . He contended that the modified radical mastoidectomy is of little value in increasing the hearing after long-standing chronic suppuration of the middle ear, because the damage that has been done to the conduction mechanism by the time operation is required is permanent.

28. McKenzie, W.: The Results of the Conservative Radical Operation or Attico-Antrotomy in Seventy Cases, J. Laryng. & Otol. **55**:75 (Feb.) 1940.

29. Maxwell, J. H., and Richter, H. J.: Radical Mastoidectomy: Its Effect on Hearing, Arch. Otolaryng. **31**:426 (March) 1940.

30. White, L. E., Jr.: Radical Operation of the Mastoid, Arch. Otolaryng. **8**:32 (July) 1928.

Maxwell and Richter [29] made the following statements:

The duration of the otorrhea bore no significant relation to preoperative loss of hearing. . . .

A relatively poor prognosis for residual hearing after a radical mastoidectomy may have to be given to persons over 45 or 50, to those in whom the infection of the middle ear and mastoid is of short duration and also perhaps to those who have comparatively good preoperative hearing as indicated by an average loss in the critical frequencies of less than 25 or 30 decibels. . . .

TABLE 26.—*Hearing Before and After Radical Operation in Relation to Otorrhea and the Healing Time of the Mastoid*

Radical Operation, Side	Hearing Before Operation	Hearing After Operation	Audiogram After Operation, Percentage Loss		Duration of Otorrhea Before Operation	Mastoid Healing Time
			Right	Left		
Right	Impaired	Slightly better	27	8	?	1½ years
Left	Impaired	Same	10	38	1½ years	1½ years
Left	Impaired	Worse	13	40	7 years	7 months
Left	Normal	Impaired	17	26	?	2 years
Right	Impaired	Slightly better	33	6	?	3 months
Left	Impaired	Worse	6	39	4 weeks	6 months
Left	Impaired	Slightly better	5	18	5 years	6 months
Left	Impaired	Better	10	60	?	2 years
Right	Impaired	Worse	48	Normal	16 years	3 months
Right	Normal	Impaired	24	13	?	?
Left	Impaired	Same	Normal	20	8 years	15 months
Right	Normal	Impaired	49	12	1 month	2½ years
Left	Impaired	Worse	17	44	10 years	2 months
Left	Normal	Impaired	29	38	13 years	Continued discharge
Left	Impaired	Worse	41	35	10 years	1 year
Right	Impaired	Same	29	27	?	?
Right	Impaired	Same	39	14	6 months	Continued discharge
Left	Impaired	Improved	12	65	3 months	9 months
Left	Impaired	Worse	Normal	38	3 months	13 months
Bilateral	Impaired	Improved	30	32	19 years	Right, 1 year; left, continued discharge
Left	Normal	Impaired	17	44	10 years	5 months
Left (modified)	Normal	Normal	13	8	1½ months	5 months
Right	Impaired	Worse	43	10	6 years	4 months
Left	Impaired	Worse	8	29	9 years	4 months
Right	Impaired	Improved	38	11	10 days	2 years
Right	Normal	Impaired	36	17	3 months	2 years
Right	Impaired	Same	26	12	10 years	3 months
Left	Impaired	Same	8	33	3 months	9 months
Left	Impaired	Same	1	37	15 years	1 year
Bilateral	Impaired	Improved	21	40	12 years (right), 1 year (left)	1 year

TABLE 27.—*Preoperative and Postoperative Hearing Loss as Shown in Audiograms in Relation to Otorrhea and Healing Time of the Mastoid*

Radical Operation, Side	Audiometric Hearing Loss, %				Duration of Otorrhea Before Operation	Healing Time of Mastoid
	Before Operation		After Operation			
	Right	Left	Right	Left		
Right	64	27	17	12	23 years	Ear still discharging
Right	14	6	32	20	? years	6 months
Left	10	47	16	28	?	6 months
Left	13	48	5	44	11 years	Ear still discharging
Left	9	33	8	29	12 years	2 months
Right	33	24	32	13	13 years	2 months
Right	84	24	50	6	1 week	6 months

Rapid healing of the wound carries a better prognosis in regard to residual hearing. . . .

In the majority of cases the chances that the hearing may be slightly improved or slightly impaired are about equal. In either event the average change is less than 10 decibels.

SUMMARY

A survey with clinical observations on 526 consecutive operations for acute and chronic mastoiditis is presented.

Mastoidectomies were done on patients of all ages, from 6 weeks to 70 years, but most frequently on patients between 4 and 12 years of age for the acute type of mastoiditis and between 19 and 30 years for the chronic type.

Operations for acute mastoiditis outnumbered the radical mastoidectomies for the chronic type by 2 to 1.

Infections of the upper and lower respiratory tract, measles and scarlet fever constituted the leading etiologic factors.

Early, adequate myringotomy was found advantageous.

Midwinter and spring were the peak seasons for acute infections of the mastoid.

Most of the operations for acute mastoiditis were performed during the second, third and fourth weeks of the infection.

The hemolytic streptococcus was the predominating organism.

Most patients with acute mastoiditis had a leukocyte count between 12,000 and 18,000.

Roentgenograms revealed a high rate of diagnostic accuracy.

No one method of postoperative treatment was found suitable for all cases in which radical mastoidectomy had been performed.

No complications developed as a result of unnecessary exposure of the dura and lateral sinuses.

Nearly 50 per cent of the patients with chronic mastoiditis had cholesteatomas. Cholesteatoma was found to be an indication for radical mastoidectomy.

There were intracranial complications in 19 per cent of the patients operated on for acute mastoiditis and in 20.2 per cent of the patients with radical mastoidectomies.

Of the 205 patients with acute surgical mastoiditis and follow-up records, 98 per cent had dry ears, and of the 123 patients with radical mastoidectomies and follow-up records, 90 per cent had dry ears.

The mortality rate for patients of all ages operated on for acute mastoiditis was 6.6 per cent, and that for patients with radical mastoidectomy was 4.5 per cent.

The residual hearing following radical operations was as good as or better than the preoperative hearing in 63.8 per cent of the 94 patients for whom there were follow-up records. The improvement apparently depended somewhat on the duration of otorrhea before operation, but was independent of the healing time.

100 West Coulter Street.

OFFICE NOISES AND THEIR EFFECT ON AUDIOMETRY

WILBER D. CURRIER, M.D.
BOSTON

There is much difference of opinion among otologists, and others who test hearing, concerning the relative value of soundproof or so-called quiet rooms for hearing tests. Some maintain that a soundproof room is necessary, while others go so far as to state that the sense of hearing, which normally is used in a noisy environment, should be tested in the presence of noise. A special committee of otologists, appointed in London in 1929 to study problems concerned with hearing tests, stated, after consideration of the testing environment, that it did "not recommend the use of a silent room for ordinary hearing tests, but that these tests should be carried out in a reasonably quiet room." [1] This conclusion, as well as others reported by the committee, was severely criticized by Hallpike [2] and others.

Since 1929 advances have been made not only in the different branches of otology, including hearing and its problems, but in the various aspects of noise [3] and of acoustics and soundproofing.[4] Today the audiometer, constructed by Dean and Bunch [5] in 1919, is used by most otolaryngologists. By means of this instrument it is possible to make accurate determinations of hearing acuity. Today, also, there are available the "reverberation meter," reported on by Sabine [6] in 1937, and the sound (noise) level meter. With these two instruments accurate measurements can be made of the acoustic value of a room. Nevertheless, as far as I know, there has been no report regarding the use of these two instruments in determining the acoustic value of hearing test rooms. Part of the study presented in this paper deals with the results I obtained in measuring the acoustic value of rooms used for hearing tests. These investigations were, however, carried out with only the sound (noise) level meter. Consequently, my results are not as accurate as they would have been had both instruments been used.

Studies relative to measurement of adventitious noises present in rooms used for hearing tests and to the effect of these noises on audiometry have also, apparently, been neglected. In a search of the literature I was unable to find any reports deal-

---

Submitted to Washington University, School of Medicine, St. Louis, as partial fulfillment of requirements for degree of Master of Science.

1. Report of Committee for the Consideration of Hearing Tests, J. Laryng. & Otol. **48**: 22 (Jan.) 1933.

2. Hallpike, C. S.: Critical Review: The Hearing Tests Committee Report, J. Laryng. & Otol. **48**:114 (Feb.) 1933.

3. (*a*) Behnke, A. R., Jr.: Noise in Relation to Hearing and Efficiency, New York State J. Med. **40**:1080 (July 15) 1940. (*b*) Carter, H. A.: Decibels and Cycles, Tr. Am. Acad. Ophth. (1940) **45**:21 (May-June) 1941. (*c*) Dennis, E. B., Jr.: Noise: Its Measurement, Effect and Control, New York State J. Med. **30**:573 (May 15) 1930. (*d*) Fowler, E. P., Jr.: Extraneous Factors in Quantitative Tests for Hearing, Acta oto-laryng. **28**:283, 1940. (*e*) McCord, C. P.; Teal, E. E., and Witheridge, W. N.: Noise and Its Effect on Human Beings; Noise Control as a By-Product of Air Conditioning, J. A. M. A. **110**:1553 (May 7) 1938.

4. Sabine, P. E.: Acoustics and Architecture, ed. 1, New York, McGray-Hill Book Company, 1932, p. 204. Watson, F. R.: Acoustics of Building, ed. 3, New York, John Wiley & Sons, Inc., 1941, p. 131.

5. Dean, S. W., and Bunch, C. C.: The Use of the Pitch Range Audiometer in Otology, Laryngoscope **29**:453 (Aug.) 1919.

6. Sabine, H. J.: Portable Reverberation Meter, Electronics, March, 1937, p. 30.

ing with these two important factors. Fletcher [7] and Fowler Jr.[3d] made significant contributions in regard to the effect of noise on hearing tests. But neither of these investigators measured his noise levels with a sound (noise) level meter. Their contributions, however, are important, and every otologist interested in the problem of hearing should review both papers.

According to Fowler Sr.,[8] "In the office or hospital, outside noises always mask the testing sounds to some extent, and commonly to as much as 15 or 20 db below the true level in a quiet place." It will be noted here that his opinion concerns the "quiet place." If this statement is true, the amount of error should be even greater than 15 to 20 decibels if tests are done in an absolutely soundproof room. Newhart [9] expressed agreement with Fowler [8] when he maintained that tests for the detection of slight, but significant, impairment in hearing are not accurate if made in the presence of interfering noises. Perhaps every person who tests hearing will agree with this. Jones and Knudsen [10] remarked:

> It is freely admitted everywhere that a careful study of a hard of hearing patient should include the vestibular tests and audiometric tests with a standardized audiometer in a soundproof booth. . . . Soundproof booths, without which any hearing test is woefully inadequate, are practically unknown.

These opinions have been substantiated by the work of Fletcher,[7] Fowler Jr.[3d] and the research which I present here. In fact, in my survey of the literature, I could find only opinions, and no investigative work, to support the idea that a soundproof environment was unnecessary for even routine hearing tests in the physician's office.

In contrast to the foregoing statements, Watson [11] maintained: "The difference between a soundproof room and a quiet office will be very, very slight—at absolute maximum about 5 decibels in the low tones." In his opinion, also, "the question of noise, the necessity of a soundproof room, has been overemphasized." Watson cited no statistics or research to support his view. It might be pointed out that Mr. Watson, who probably uses the audiometer chiefly for the fitting of hearing aids, usually tests patients with rather severe deafness. A noise of 30 decibels will not interfere in the testing of a person with a loss of 50 decibels or more. In my hands the audiometer is not used one time in fifty for the purpose of fitting a hearing aid. It is employed principally for the detection of early hearing loss, for arriving at a diagnosis, for determining the progress of the hearing loss, for checking results of therapy and for estimating the prognosis. In all instances in which such information is desired the hearing loss usually is near the normal threshold, or the "zero line," and small losses or gains (10 to 20 decibels) in hearing are important.

It is of interest to note that, almost without exception, the otologist in each office in which the investigations reported here were conducted apparently believed that his testing room was quiet. At least, nearly every one stated that his room was "entirely satisfactory" and that his results were "sufficiently accurate."

---

7. Fletcher, H.: Speech and Hearing, New York, The Macmillan Coompany, 1929.

8. Fowler, E. P., Sr.: Medicine of the Ear, New York, Thos. Nelson & Sons, 1939, p. 369.

9. Newhart, H.: Progress in the Conservation of Hearing, Ann. Otol., Rhin. & Laryng. **50**:129 (March) 1941.

10. Jones, I. H., and Knudsen, V. O.: What Audiometry Can Now Mean in Routine Practice, J. A. M. A. **111**:597 (Aug. 13) 1938.

11. Watson, L. A.: Audiometers, Hearing Aids, and the Ear Specialist: An Address and Symposium, Minneapolis, Minn., The Maico Company, Inc., 1941, p. 15.

Nevertheless, the noise meter records and the audiometric readings secured under the circumstances which customarily prevailed in the testing room of each otologist visited proved otherwise.

Of course, it is not impossible to do acceptable, or even good, work in the presence of some noise. The noise level should, however, be constant, and the only constant noise level is obtained in a soundproof room. Sjöberg [12] stated that Holmgren used an electric fan in his soundproof room as a constant source of adventitious noise. If Holmgren had checked his electric fan, the noise of which "sounded" so constant, he would have found that there was a notable fluctuation in the noise level. Besides, if he really had a soundproof room, he had no need to seek further for a constant, controlled and ideal noise level.

Larsen [13] produced valuable research on occupational deafness, and he used only one of his office rooms for the hearing tests. Concerning the matter of extrinsic noises in his office, he remarked that "although the clinic is situated in a main thoroughfare, it has been sufficiently quiet, because the examinations were generally made in the evening." Possibly thoroughfares in Sweden are more quiet than those in the United States. My findings indicate that street noise is one of the most important causes of inaccuracy in the results of hearing tests. Mr. Dennis,[3c] of the Noise Abatement Commission of New York city, reported an average of 81 decibels of noise on busy streets of that city.

Today the most significant advance in otology is, perhaps, the early recognition and treatment of deafness, especially in children. The investigations of Crowe and Burnam [14] and Guild and associates,[15] in Baltimore, and of Gardner,[16] of the department of health in Oregon, are particularly outstanding. Hearing losses, when found early, are usually not great. But accurate and reliable measurements of such hearing losses are impossible to obtain without a noiseless testing environment. Excellent research and clinical investigations of many workers have been criticized because the workers neglected to conduct their studies in a noiseless environment.

In determinations of the result of therapy for deafness, Rosenthal [17] observed that when the hearing loss is in the "medium range" an improvement of even 5 decibels will elicit a subjective response. His tests, however, were made not in a soundproof room but "in rooms adjoining the clinic or office," and "there was an ordinary amount of noise from adjacent activities." Several of my most inaccurate results were obtained in the presence of similar adventitious noises. For measuring of small losses or gains in hearing, especially if they are in the "medium range" or near the threshold, a soundproof testing room is necessary before accurate results can be obtained. This is especially true when one is checking the results of therapy, in which case changes in hearing frequently are small. Hughson and Witting [18]

12. Sjöberg, A. A.: Critical Review of Electro-Acoustic Test Methods and Choice of Amplifier to Improve Hearing, Acta oto-laryng. **28**:437, 1940.

13. Larsen, B.: Investigations of Professional Deafness in Shipyard and Machine Factory Labourers, Acta oto-laryng., 1939, supp. 36, pp. 3-255.

14. Crowe, S. J., and Burnam, C. F.: Recognition, Treatment and Prevention of Hearing Impairment in Children, Ann. Otol., Rhin. & Laryng. **50**:15 (March) 1941.

15. Guild, S. R.; Polvogt, L. M.; Sandstead, H. R.; Loch, W. E.; Langer, E.; Robbins, M. H., and Parr, W. A.: Impaired Hearing in School Children, Laryngoscope **50**:731 (Aug.) 1940.

16. Gardner, W. H.: Report of the Committee on Hard of Hearing Children for the School Year of 1938-1939, Tr. Am. Soc. Hard of Hearing, 1940, p. 103.

17. Rosenthal, M.: Use of Prostigmine for Impaired Hearing, Arch. Otolaryng. **34**:540 (Sept.) 1941.

18. Hughson, W., and Witting, E. G.: Estimation of Improvement in Hearing Following Therapy of Deafness, Ann. Otol., Rhin. & Laryng. **49**:368 (June) 1940.

maintained that 10 decibels should be the amount of minimum improvement that can be of real significance, and this only if originally obtained levels can be improved by such a figure. The required duration of improvement has been set at five years. It certainly would be difficult to compare the noise levels in a so-called quiet office after five years had elapsed.

It has been demonstrated many times, and again recently by Kinney,[19] that hearing for voice sounds is from about 5 to 15 decibels less acute than hearing for the pure tones of the audiometer. Hence, as Fowler Jr.[3d] showed, under identical conditions noises would interfere more with the testing of voice comprehension than with the testing of hearing of audiometric tones. The otologists, therefore, who

TABLE 1.—*Intensities of Noise in Common Environments* *

Source or Kind of Noise	Source of Determinations: Currier (Offices)	McCord [3e]	Fletcher [7]	Sutherland [22]	Electronics [23]	Approximate Average
Threshold of hearing	..	0	..	0	0	0
Soundproof booth	<24	..	0	..	..	0 (?)
Rustle of leaves	..	10	..	15	10	10
Acoustically designed office	25	..	..	30	..	30
Average whisper	..	20	..	25	15	20
Country road (garden)	..	..	..	30	20	25
Average dwelling	..	..	..	35	25	30
Quiet office	30	..	20	40	30	30
Noisy office	40	..	40	..	..	40
Average office	35	..	30	50	35	40
Train or automobile	..	..	50	..	45	50
Conversation	..	50	..	60	55	55
Typewriter	40	..	..	70	..	55
Radio in home	..	65	..	45	..	55
Business street	..	75	..	65	65	70
Subway train	..	..	60	85	..	75
Streetcar	35	80	..	..	..	60
Police whistle	35	80	..	..	..	60
Heavy street traffic	..	85	..	90	80	85
Express train	..	105	..	75	..	90
Elevated train	..	..	..	95	90	90
Thunder	..	70	..	..	120	95
Pneumatic drill	..	90	..	130	85	100
Riveter	..	..	..	105	100	105
Boiler factory	..	..	80	110	100	100
Automobile horn	..	100	..	115	...	105
Airplane engine	..	115	..	120	110	115
Threshold painful sound	..	120	..	...	...	120
Sound representing limit of endurance	..	130	..	...	...	130

* All figures represent decibels.

still use only voice testing, and those who use voice testing as an adjunct to audiometry (as is proper), should take into consideration the fact that noises will affect voice testing even more than they will audiometric examinations. This point should also be borne in mind by those who use the phonograph in group testing in the schoolroom or in other "noisy" places.

Witting and Hughson,[20] Bunch [21] and others have shown that the normal range of error in audiograms is ± 5 decibels. To obtain repeated tests within this range

19. Kinney, C. E.: Testing Hearing and Evaluation of Results in Mathematical Figures, West Virginia M. J. **37**:448 (Oct.) 1941.

20. Witting, E. G., and Hughson, W.: Inherent Accuracy of a Series of Repeated Clinical Audiograms, Laryngoscope **50**:259 (March) 1940.

21. Bunch, C. C.: Personal communication to the author.

of error, it is necessary that each test be made in a soundproof chamber by a competent operator and that the same operator perform all the tests. If under such relatively ideal conditions there is a range of error of 10 decibels (± 5), what must the error be when 30 to 40 decibels of noise (which is common) is thrown on the ears of the patient being tested? Fowler Jr.[3d] found that an estimated masking noise of 25 decibels had a deafening effect of 35 decibels.

In table 1 are listed the noise levels present in various common environments, as determined by Fletcher,[7] by Sutherland,[22] by McCord and associates,[3e] by *Electronics* [23] and by me. It will be noted that the data given by these investigators vary somewhat, surprisingly so for some noise sources. My experience with determinations of noise levels has impressed me with the generalization necessary to arrive at figures such as are given in table 1. I had difficulty in determining a "general noise level" because the sounds responsible for the noise were not at all constant even when they seemed so to the ear. Widely varying noise intensities, from almost zero to often 70 to 80 decibels, were always present in the offices of practically all the physicians visited. The indicating needle on the noise meter was in almost continual fluctuation. Apparently, under some conditions one's ears become so accustomed to sharply changing sound intensities that one has in a large measure learned to disregard them. One merely raises one's voice and adjusts one's actions to compensate for the noises, without being aware of doing so.

As Behnke [3a] pointed out, "Rattling, squeaking or intermittent or erratic sounds may cause an annoyance out of all proportion to their intensity." Thus, an automobile horn or a policeman's whistle is an especially pernicious source of noise. The Noise Abatement Commission of New York city [3c] found that the degree of annoyance of a noise depended on several factors, such as its intensity, its component frequencies, its general character (steady or intermittent) and whether or not it was regarded as necessary. The patient does not want, nor does he expect, noises to interfere with his hearing test. He is, or should be, concentrating intently on the test he is taking. Intermittent, and even slight, noise, which is overlooked by the examiner, may, therefore, be distracting to the patient. Bunch [21] noted this fact clinically. It is well known that one of the greatest sources of error in audiometric examinations is the patient's failure to give undivided attention to his test. His distraction is frequently due to the presence of an interfering noise.

On the other hand, every one who makes hearing tests has probably observed how unduly impressed some patients are with the quietness of the testing room. Often the patient will even remark that the room is "very soundproof." Apparently, most otologists are equally impressed with the quietness in their testing rooms.

PRESENT INVESTIGATION

My associate, Dr. Frank Impastato,[24] helped me conduct some of the investigations which are reported here. Our investigations were carried out in 15 hearing test rooms in or near St. Louis. Eleven were made in the testing rooms of practicing otolaryngologists and 4 in schools (1 grade school, 1 high school and 2 universities, 1 of which, the Washington University School of Medicine, had a soundproof

22. Sutherland, R. T.: The Noise Problem and Its Minimization Aboard Ship (unpublished report); cited by Behnke.[3a]

23. Electronics' Chart of Sound Levels, Electronics **4**:43 (Feb.) 1932.

24. Without the industry, cooperation, and stimulation of Dr. Frank Impastato the present work could not have been done. Both he and I had just completed a year of postgraduate study and had had special training in audiometry under Dr. C. C. Bunch when this research was undertaken.

room). One of the most inaccurate hearing tests (because of noise) was obtained in the university which did not have a soundproof room. Our investigations were conducted in the following manner:

1. Dr. Impastato and I made all the audiometric tests, in all of the testing rooms, on one another. Twelve audiograms, 6 for each of us, were taken in the soundproof room of the Washington University School of Medicine. These 12 audiograms were averaged, and the average arrived at was used as the normal hearing threshold for each of us. The audiometric readings taken in the remaining fourteen rooms were compared with this "normal" reading.

2. In each office we visited we asked the office personnel to permit us to work under the identical environment that prevailed during their routine tests. In all tests we conducted, we used the Western Electric 2A audiometer, which was checked several times during the research against the Western Electric 1A and the Maico audiometer. A General Radio Company sound level meter, type 759A, was used for measurement of the noises present in the hearing test rooms. Unfortunately, this instrument records only noise intensities of 24 decibels or above.

In spite of the fact that the noise level meter did not record noise intensities below 24 decibels, valuable results were obtained. Every room in which tests were made, including the soundproof room at Washington University, permitted noises to enter which could be heard, but which were of an intensity too low to be indicated on the noise meter. It was noted, however, that a noise had to be extremely loud in order to come through even faintly, perhaps at 5 to 10 decibels, into the soundproof room at Washington University. In all the other rooms many sounds came from the outside besides those which were recorded. These unrecorded sounds were heard distinctly during the hearing tests, but were of less intensity than 24 decibels, so that they could not be measured.

Many other interesting observations were made during the visits to the offices of the various otologists. In 7 offices the physician himself took the audiograms. In the remaining 4 offices and in the schools a nurse, an office girl or a technician made the test. In 1 of the universities "just any one" made the test. The testing methods and the technics used varied to unbelievable degrees. As a result, comparison of audiometric records obtained in one office with those obtained in another, irrespective of the vast differences in noise, gave a discouraging impression. I was convinced that some acceptable standardized technic for audiometric tests should be formulated and presented to members of the medical profession. Valuable suggestions regarding the technic of making audiometric tests can be found in the excellent and timely text on audiometry by Bunch.[25]

Only in 5 of the 15 rooms tested (including the soundproof room) was there any pretense whatever at soundproofing. The remaining 10 rooms were ordinary treatment rooms, storage rooms or small clothes closets (3 by 4 feet [0.9 by 1.2 meters]), into which extraneous noises could easily enter. Yet in the face of this, and other, factors, almost all the otologists insisted that they had a "good, quiet room" and were getting "good results." In addition, some of these rooms were uncomfortably hot and stuffy. Many rooms had squeaky chairs or other noisy equipment. One indicator button squeaked to 35 decibels each time it was pushed. In table 2 are shown the approximate amounts of noise that entered the rooms in which the investigations were carried out. The determinations of the noise levels, with the noise meter, were made with the doors of the testing rooms open, as well as closed. All noises originating both inside and outside the testing rooms were recorded. The otologists who watched the noise meter in operation in the rooms they routinely used for audiometric tests were amazed at the amount of noise interference that could be demonstrated.

25. Bunch, C. C.: Clinical Aspects of Audiometry, to be published.

The audiograms my associate and I took on each other in the soundproof room were used as a standard, or normal, with which the results of all the other audiometric tests we made were compared. One of the first questions which might be raised is: "How accurate were the audiograms which you took on each other in the soundproof room?" The accuracy of our audiometric tests was established on the basis of conclusions drawn by Witting and Hughson [20] from their excellent research on the inherent accuracy of audiograms. Their testing conditions and

TABLE 2.—*Noises in Testing Rooms of Various Otologists* *

Source of Noise	Most Quiet Testing Room		Most Noisy Testing Room		Approximate Average Noise Level of All Testing Rooms	
	Doors Open	Doors Closed	Doors Open	Doors Closed	Doors Open	Doors Closed
In outer office, etc.:						
General noise level	25-30	<24	30-40	25	35	<24
Talking	25-35	<24	30-40	25-30	35	<24
Walking	30-35	25-30	35-40	30-40	40	30
Opening and closing of doors	40-50	25-40	40-50	30-40	45	35
Clicking of instruments	40-50	<24	..	..	45	35
Typing	30-40	<24	35-40	30	35	25
Rustle of papers	30-35	25-30	30-35	25-30	35	25
Office buzzer	..	..	30	25	30	25
Noises in adjoining offices, hall or elevator	35-50	35-40	35-50	25-40	45	35
Automobile horns	35-45	<24	40-50	40	45	35
Police whistle	35	<24	..	..	35	25
Streetcars	30	<24	..	..	35	25
In testing room:						
Electric fan	30-35	33-38	..	..	40	40
Running water	..	..	30	30	30	30
Squeaking of metal stool	30-40	30-40	..	30-40	..	35
Coughing	..	35-40	..	35-40	..	40
Rubbing of hands briskly	..	30	..	30	..	30
Rubbing of clothing	..	25	..	25	..	25
Squeaking of shoes	..	25-30	..	25-30	..	30
Clearing of throat	..	40	..	40	..	40
Snapping of light off and on	..	40-50	..	40-50	..	45

* All figures represent decibels.

TABLE 3.—*Comparative Accuracy of Audiograms*

Decibel Range	Witting and Hughson [20] Soundproof Room (10 Tests on 17 Persons)	Present Study *					
		Office Testing Rooms (14 Rooms; 14 Tests, 1 in Each Room)		Soundproof Room (12 Tests)		Increased Accuracy in Soundproof Room	
		Dr. A	Dr. B	Dr. A	Dr. B	Dr. A	Dr. B
±5	93.1%	76.9%	81.2%	98.8%	95.8%	21.9%	14.6%
±2.5	62.1%	61.1%	66.3%	90.6%	81.2%	29.5%	20.1%

* Percentages calculated after the manner described by Witting and Hughson.[20]

environment were essentially similar to ours, except that they took not less than 10 audiometric readings on each patient and we took but 6 on each other (12 in all). These investigators observed that the average of 3 audiograms reveals a much smaller error than that shown by a single test, and recommended such a procedure whenever accuracy is desired.

Witting and Hughson [20] found that 93.1 per cent of all tones in their most accurate records were within the accepted ± 5 decibel range on repeated tests; 62.1 per cent of the tones in their records were within ± 2.5 decibels. Our results were as follows: In the audiograms taken on Dr. A, 98.8 per cent of all tones were

within a ± 5 decibel range, and 90.6 per cent were within a ± 2.5 decibel range. In the audograms taken on Dr. B, 95.8 per cent were within a ± 5 decibel range and 81.2 per cent within a ± 2.5 decibel range. Why our results appear to be somewhat better than those of Witting and Hughson I cannot say.

In table 3 the accuracy of the audiometric results obtained in our soundproof room are compared with those obtained in the other testing rooms we visited. It will be noted that in the soundproof room Dr. A was 22 per cent more accurate for the ± 5 decibel range and 30 per cent more accurate for the ± 2.5 decibel range. Dr. B, in the same room, was 15 per cent more accurate for the ± 5 decibel range and 20 per cent more accurate for the ± 2.5 decibel range. When the results

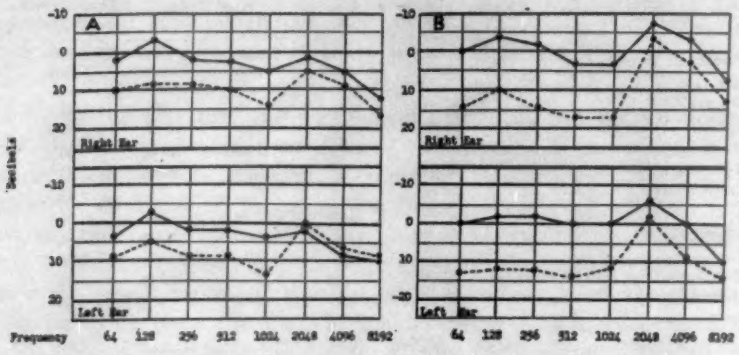


Chart 1.—Comparison of audiograms taken in a soundproof room (solid line) and in a physician's testing room (line of dashes) for (*A*) Dr. A. and (*B*) Dr. B.

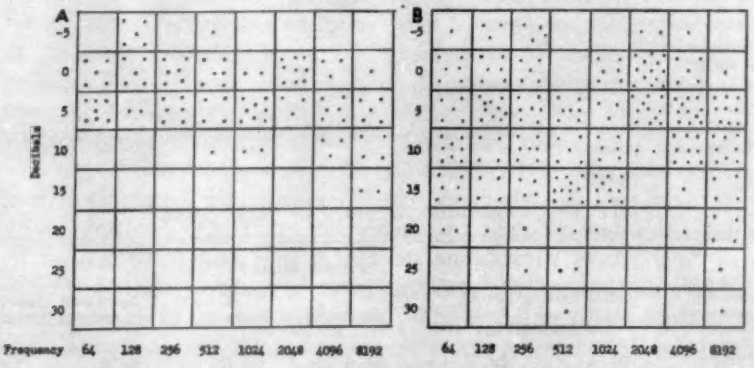


Chart 2.—Scattergrams showing distribution of tones in all tests on Dr. A. in (*A*) a soundproof room and in (*B*) a physician's testing room.

obtained by both Dr. A and Dr. B are averaged, it is found that there was 18.4 per cent greater accuracy for the ± 5 decibel range and 22.7 per cent greater accuracy for the ± 2.5 decibel range in the soundproof room than in the other testing rooms.

After a review of the inaccuracy of the records taken in the testing rooms of various physicians, as shown in table 3, the "averaged" audiograms (chart 1 *A* and *B*) are surprising on first consideration. One would probably expect a greater error to be demonstrated in such audiograms. However, the following facts must be kept in mind: First, only 1 of the 15 testing rooms was com-

pletely soundproofed. Four others were fairly well soundproofed. These 4 rooms helped a great deal to bring up the average accuracy. In fact, the records obtained in these 4 rooms were nearly identical with those obtained in the soundproof room. Second, the tone thresholds obtained in the various offices were so scattered that it had to be concluded that the accuracy of single audiograms taken in noisy environments is unreliable. The scattergrams (chart 2*C* and *D*) demonstrate the wide variance of records on comparison.

Another instructive comparison is that which is shown in chart 3. The audiograms in this chart demonstrate the effect of noise in the testing room of one of the schools visited. They also demonstrate the well kown fact that noise in a hearing test room affects the low tones but has little effect on the high tones. It is because of this masking effect of noise on low tones that some otologists formerly believed that conductive deafness showed a greater loss for low tones than for high tones. They were merely testing in the presence of noise, without realizing its effects.

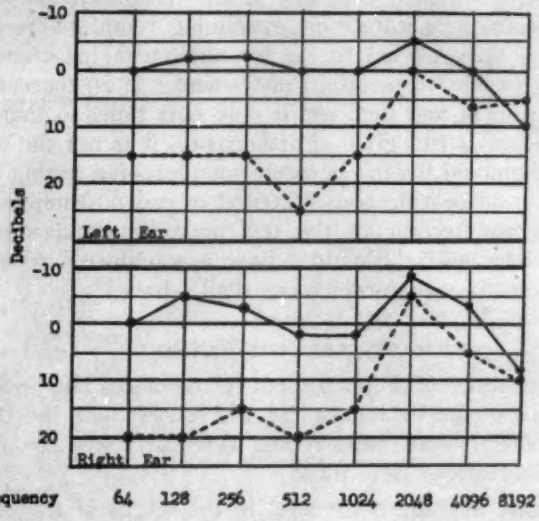


Chart 3.—Comparison of audiograms for Dr. B. taken in a soundproof room (solid line) and in noisy testing room.

COMMENT

Only in the past few years has the otologist become awakened to two important realizations. First, he must become skilled in the fitting of hearing aids. As the deafness of the patient who requires a hearing aid is usually severe, his audiometric test could, possibly, be satisfactorily made in a reasonably quiet room. The second realization, and the one which is vastly more important, is that diagnosis of deafness, particularly in children, must be made early and treatment instituted without delay. For this purpose a soundproof environment is necessary. The otologist today knows that the outstanding preventive measures for progressive deafness are intelligent observation of the patient and early repeated testing of his hearing. From statistical studies, Fowler Sr.[8] estimated that one third of all school children show a loss of at least 15 decibels at some frequency. Up to 8 or 10 years of age, deafness is apt to be unilateral, and so usually goes unnoticed. The United States Department of Health reported that there are 500,000 children who need special instruction in hearing, as compared with 66,000 children who need special instruction because of defective vision.

Many state departments of health are doing excellent work in both group and individual testing of the hearing of school children. Because of the testing environments in which they are usually forced to work, their tests are, however, at times somewhat inaccurate. Nevertheless, often they are more accurate than the tests made by the local otologist, who is, and should be, the last court of appeal to persons seeking accurate hearing tests.

Most otologists believe that a soundproof room is too expensive, that it requires too much space and that it is not practical. Actually, this is not the case. The Medical Center, New York, built a soundproof room of celotex for a cost of $60. Newhart and Hartig,[26] as well as Jones and Knudsen,[10] gave details for constructing a small, portable booth (4 by 4 feet [1.2 by 1.2 meters]) that will fit into an office room. Such a room is satisfactory for routine office audiometric tests. More expensive and more elaborate rooms have been described in detail by Burr and Mortimer [27] and by Bunch.[25]

At best, hearing tests are time consuming. The test is further prolonged and valuable time is wasted when the otologist or the technician has to stop the test each time an extraneous noise enters his examining room. This probably has a tendency to make the otologist fail to use his audiometer as often as he should. An audiometer is an expensive precision instrument. It is, therefore, not reasonable to buy this instrument and then use it only part time, so that one gets from it less than the efficiency it can give. Furthermore, it is not the audiometer but, rather, the faulty technic and the inadequately soundproofed testing rooms, that are responsible for inaccurate hearing tests. Hence, in order to improve the accuracy of audiometric tests, the technic of the test must be standardized. Also, the otologist must no longer ask: "Should I have a soundproof room?" Today he must ask: "What kind of soundproof room shall I have?"

SUMMARY AND CONCLUSIONS

A survey has been made of noises present in the offices and audiometric testing rooms of 11 practicing otolaryngologists and of 4 schools. Of the 15 rooms visited, only 1 was soundproofed. Four other rooms were fairly well soundproofed.

The following observations were made:

1. The approximate average noise level in the offices of the otolaryngologists was 35 decibels.

2. The approximate average noise level in the hearing test rooms of the otolaryngologists, under audiometric testing conditions, was less than 24 decibels.

3. The audiograms taken in the testing rooms of the otolaryngologists were approximately 20 per cent less accurate than the audiograms taken in the soundproofed room of the university.

4. The noise level in the offices, as demonstrated by audiometric tests, caused a lowering of the hearing threshold by approximately 10 to 15 decibels.

5. Noises in the testing room as shown in 1 instance in the present study lowered the hearing threshold by 20 to 25 decibels.

6. Noises present in the testing room affected chiefly hearing for the accuracy of the tests for low tones (below 2048 double vibrations), hearing for high tones being but little affected.

---

26. Newhart, H., and Hartig, H. E.: An Efficient and Practical Booth for Testing Hearing, Arch. Otolaryng. **28**:1000 (Dec.) 1938.

27. Burr, E. G., and Mortimer, H.: Improvements in Audiometry at the Montreal General Hospital, Canad. M. A. J. **40**:22 (Jan.) 1939.

7. The threshold tones obtained in the average testing room varied widely in comparison with those obtained in the soundproof room.

From the data obtained, the following conclusions may be drawn:

1. Unless the hearing test room is soundproofed, the extraneous office noises will significantly decrease the accuracy of routine audiometric tests.

2. The technic used for audiometric examinations varies greatly in different offices. As a result, the accuracy of routine tests is significantly decreased, and reliable comparison of records from different offices is prevented.

3. Audiograms taken by different operators, using various technics, show such a wide variation that their accuracy cannot be accepted. The results of such tests can be used only as rough estimates.

4. The majority of otologists do not have rooms specially designed for hearing tests. Most of them use an ordinary treatment room, a storage room or a cloakroom.

5. Most otologists who do not have soundproofed rooms believe their testing rooms are adequately quiet for audiometric examinations and that their records are sufficiently accurate.

395 Commonwealth Avenue.

Case Reports

PRESENCE OF CHICKEN BONE IN ESOPHAGUS FOR FIFTY-SIX DAYS

D. F. Weaver, M.D., Detroit

Moersch [1] stated: "While it is true that bone tends to perforate the esophageal wall rather rapidly, it may remain in the esophagus a long time without producing a perforation." Vinson [2] stated: "The esophagus is particularly intolerant to the presence of a foreign body, especially of bones."

It is generally understood that a foreign body in the esophagus is likely to perforate the wall rather readily. Bone apparently has more of a tendency to perforate than a metallic foreign body. It is felt that the following case is of interest since the foreign body, a chicken bone, was present for a relatively long time without producing dangerous complications.

REPORT OF A CASE

A white man aged 39 was admitted to the Henry Ford Hospital on Oct. 9, 1942. He stated that on Aug. 15, 1942, while eating hash, he had swallowed a sharp object, which seemed to lodge at the lower end of his "breast bone."

There was slight, constant, dull pain in the region of the epigastrium, which became more severe when he attempted to swallow solid food or when he was in motion. There had been no hematemesis, but he had vomited when attempting to swallow solids. He was able to swallow warm liquids with little difficulty.

Roentgenograms taken by his local physician about a week after the onset of the present illness were reported as showing nothing significant.

There had been a loss of weight of 20 pounds (9.1 Kg.).

During the several weeks before admission the pain had become less severe, but the swallowing of liquids had become more difficult.

No significant abnormalities were noted on physical examination. The patient was edentulous. The blood pressure was 132 systolic and 80 diastolic. The pulse rate was 70 per minute and the respiratory rate 20 per minute. The temperature was 98 F. The hemoglobin was 13.5 mg. per hundred cubic centimeters; the red cells numbered 4,840,000 and the white cells 8,500, with 60 per cent polymorphonuclear neutrophils, 5 per cent eosinophils and 35 per cent small lymphocytes. The result of the Kline exclusion test was negative.

Urinalysis showed a specific gravity of 1.030, and acid reaction and no albumin and sugar; a few pus cells were present.

The roentgenologic report was as follows: "There is a partially obstructing lesion at the juncture of the upper and the middle third of the esophagus, with some residual food fragments lying above this point" (fig. 1 *A*).

On October 12, with local anesthesia, esophagoscopy was carried out. At the juncture of the upper and the middle third of the esophagus a bone was readily seen. Moderate esophagitis was present. The bone was removed without difficulty (fig. 2). Moderate bleeding was noted.

The postoperative course was uneventful, and on October 15 the patient was dismissed from the hospital completely free of symptoms. There was no elevation of temperature during the hospitalization.

On the day of dismissal the roentgenologic report was as follows: "There is no present evidence of the lesion previously reported in the esophagus. There is no obstruction to the passage of barium sulfate" (fig. 1 *B*).

The patient has remained completely free of symptoms for three months.

Henry Ford Hospital.

From the Division of Otolaryngology, Henry Ford Hospital.

1. Moersch, H. J.: Personal communication to the author.

2. Vinson, P. P.: Foreign Bodies in the Air and Food Passages, M. Clin. North America **16**:1471, 1933.

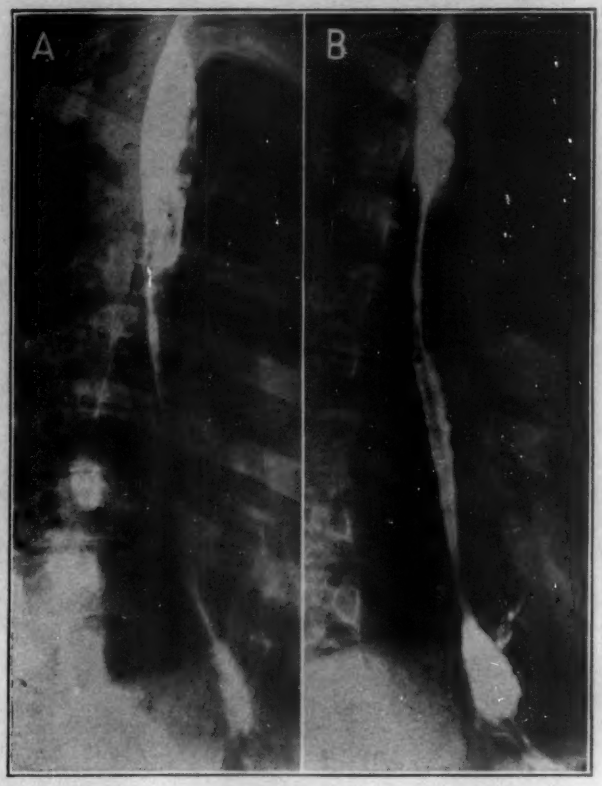


Fig. 1.—Roentgenograms of the esophagus, taken (*A*) with the obstructive lesion and (*B*) after removal of the bone.

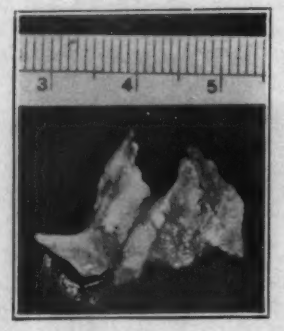


Fig. 2.—Photograph of the bone removed from the esophagus.

NEURILEMMOMA OF THE NASAL SEPTUM

R. M. Bogdasarian, M.D., and Arthur Purdy Stout, M.D., New York

Owing to the extreme rarity of tumors of the nerve sheath in the intranasal region, it seems of interest to report a case of such a growth.

REPORT OF A CASE

J. F., a white man aged 42, a Greek, was admitted to the urologic ward of the Presbyterian Hospital on Dec. 4, 1942 with a diagnosis of renal calculus. Because of the complaint of difficulty in breathing through the nose, with persistent obstruction, consultation with an otorhinolaryngologist was requested. The patient stated that five years previously he had been struck on the nose by the recoil of his gun, with resulting profuse epistaxis of two hours' duration. About two years previous to the present admission he began to notice difficulty in breathing through the nose, and about one and a half years before he felt an obstruction in the left side of his nose. There was no associated pain. On a previous admission to the hospital (subsequent to the injury), in 1937, no evidence of nasal obstruction other than enlarged inferior turbinates was noted.

The past and family histories were irrelevant. He did not smoke.

On examination the patient appeared healthy, well developed and well nourished. Intranasal inspection disclosed complete obstruction of the left nasal passage, beginning about 1 cm. from the columella, by a firm, ovoid, pale reddish pink, nontender mass, attached to the nasal septum at Kesselbach's area. The rest of the otolaryngologic examination revealed nothing abnormal.

Laboratory studies revealed a negative Kline reaction of the blood, 15.7 Gm. of hemoglobin per hundred cubic centimeters (Tallqvist), a white blood cell count of 13,250 per cubic millimeter and albumin in the urine.

With topical anesthesia induced with 10 per cent cocaine hydrochloride the mass was easily removed by a nasal snare, an oval area of nasal cartilage 2.5 mm. in diameter being exposed. The edges of the mucous membrane were touched with silver nitrate stick. Gross examination presented a firm, reddish pink, ovoid mass, measuring 1.5 by 1.3 by 0.8 cm. The specimen was fixed in Bouin's fluid.

Histopathologic Examination (figure).—The tumor was sharply circumscribed but not actually encapsulated. The superficial surface was covered with a thin mucosa, which rested directly on the tumor, and the deep surface was incompletely outlined by a very thin layer of fibrous tissue, which may have been the perichondrium. The growth was made up of two types of tissue, corresponding to Antoni's type A and type B.[1] The two were patchily intermingled and sharply defined one from the other, with type A tissue predominating. The latter was composed of twisted bands of elongated schwannian cells, with their nuclei alined in palisade arrangement, giving them a distinct organoid appearance. This tissue joined the loose-textured type B tissue, which showed microcystic degeneration. The vessels were inconspicuous and did not have thickened sheaths. The morphologic picture was typical of a neurilemmoma.

Diagnosis.—The diagnosis was neurilemmoma of the nasal cavity (septum).

COMMENT

This tumor has been called by such names as neurilemmoma, neurinoma, peripheral glioma, schwannoglioma and schwannoma. A review of the literature disclosed no case of a tumor of the nerve sheath on the nasal septum. Terplan and Rudofsky [2]

From the Department of Otolaryngology, the Presbyterian Hospital, and the Surgical Pathology Laboratory of Columbia University, College of Physicians and Surgeons.

1. Stout, A. P.: Am. J. Cancer **24**:751, 1935.

2. Terplan, K., and Rudofsky, F.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **14**:260, 1926; cited by Mittelbach and Woletz.[3]

reported a case of neurinoma of the left nasal cavity and ethmoid sinuses in a 13 year old girl. Mittelbach and Woletz [3] reported 2 cases of neurinoma, one tumor occurring in a man aged 25, involving the antrum and ethmoid sinuses on the left side, and the other in a youth aged 17 years, involving the nasal mucosa of the right middle meatus.

Tumor tissue of type A, showing nuclear palisading situated immediately beneath the mucosa.

Cruveilhier [4] reported a probable case in which the tumor sprang from the second branch of the trigeminus nerve, passed through the sphenopalatine foramen and projected into the nasal cavity, where it was mistaken for a polyp.

3. Mittelbach, M., and Woletz, F.: Med. Klin. **31**:275, 1935.

4. Cruveilhier, J.: Traité d'anatomie pathologique générale, Paris, J.-B. Baillière & fils, 1849-1864, vol. 3, p. 622; cited by Virchow, R.: Die krankhaften Geschwülste, Berlin, A. Hirschwald, 1863, vol. 3, p. 298.

Stout [1] described such a tumor in the sheath of a small nerve twig in the wall of the antrum and another in the nasopharynx.

Another probable neurilemmoma of the antrum, springing from a small nerve in the wall, was reported by Weinhold.[5]

SUMMARY

A case of neurilemmoma of the nasal septum is reported, with a review of the literature of cases of similar tumors, involving the nasal cavity.

Presbyterian Hospital.
630 West One Hundred and Sixty-Eighth Street.

5. Weinhold, C. A.: Ideen über die abnormen Metamorphosen der Highmorshöhle, Leipzig, W. Rein, 1810, p. 188; cited by Virchow, R.: Die krankhaften Geschwülste, Berlin, A. Hirschwald, 1863, vol. 3, p. 298.

AN UNUSUAL NASAL TUMOR

BERT E. ELLIS, M.D., INDIANAPOLIS

Adenocarcinoma is a malignant tumor which grows from any mucosa in which the epithelium is cylindric and from glands and ducts having cylindric cells in their lining. It might truly be called cylindric cell cancer. It is the malignant counterpart of benign adenoma.

Adenocarcinoma is not common in the nasal cavity and the accessory sinuses. New [1] found only 6 adenocarcinomas in a series of 91 primary tumors of the antrum. Fabricant [2] reported only 1 case of adenocarcinoma in 23 cases of nasal tumor. Nash [3] noted none in a smaller series, but Hill [4] found 2 adenocarcinomas among 8 malignant tumors, both in the antrum.

Ringertz [5] reported 10 cases of intranasal adenocarcinoma in a large series of cases of malignant tumors of this region from Radiumhemmet and Sabbatsberg's Hospital, Stockholm. In practically all the cases of adenocarcinoma reported in the United States, the antrum has been the site of the tumor. Ringertz, however, contended that the anterior ethmoid cells and the superior part of the nasal cavity is the site of origin of practically all such tumors. He stated with respect to this point:

> The previously published adenocarcinomas, which judging from the descriptions were really such and not solid cylindric cell carcinomas or mixed salivary tumors, have, as far as I can discover, been always localized in the superior section of the maxillo-ethmoidal region.

The reports in this country are not in agreement with those of Ringertz in this respect. New, of the Mayo Clinic, in a personal communication, stated that a review of the files at that institution revealed 50 cases of adenocarcinoma of the antrum and only 25 cases of the intranasal type. He also reported several cases of cure, in which electrocoagulation and radium were used. Part of this variation in observations may be due to differences in pathologic classification. Furthermore, Ringertz did not describe the method of treatment used in any detail. I have been unable to find any mention of metastasis in the American literature, though most reports do not discuss the subject. Correspondence with several men who have reported such tumors indicated that occasional metastasis has occurred in cases in which the tumor was not completely eradicated at operation.

It must be remembered, however, that this tumor rapidly spreads by direct extension to surrounding sinuses. A case has been reported in which the tumor extended to the external auditory canal. It may be seen, therefore, that the exact point of origin cannot always be determined. In the case which I am reporting the tumor arose from the anterior ethmoid area; however, in a previous case encountered in my practice in which the diagnosis was adenocarcinoma the origin was the antrum. It must not be assumed that nasal adenocarcinoma does not

---

From the University Medical Center, Indiana University.

Read at the meeting of the Middle Section of the American Laryngological, Rhinological and Otological Soc., Inc., Detroit, Jan. 20, 1943.

1. New, G. B.: Malignant Diseases of the Paranasal Sinuses, Am. J. Surg. **42**:170 (Oct.) 1938; Malignant Tumors of the Antrum of Highmore, Arch. Otolaryng. **4**:201 (Sept.) 1926.

2. Fabricant, N. D.: Incidence of Malignant Tumors of Head and Neck, Arch. Otolaryng. **29**:65 (Jan.) 1939.

3. Nash, C. S.: Management of Malignancies of the Sinuses, Ann. Otol., Rhin. & Laryng. **44**:220 (March) 1935.

4. Hill, F. T.: Malignant Disease of the Sinuses, Ann. Otol., Rhin. & Laryng. **46**:158 (March) 1937.

5. Ringertz, N.: Pathology of Malignant Tumors Arising in the Nasal and Paranasal Cavities and Maxilla, Acta oto-laryng., 1938, supp. 27, p. 1.

result in fatalities because it seldom metastasizes. Death usually occurs by extension of the tumor to the base of the brain, followed by fatal meningitis.

In most reports in this country these tumors have not been subdivided, but have been mentioned only as adenocarcinoma grade 1, 2, 3 and 4. Ringertz, however, expressed the belief that the nasal adenocarcinomas fall into two types: the alveolar and the pseudopapillary.

A tumor of the alveolar type has essentially the same construction as the adenoma except for the greater cell atypia and the number of mitotic figures. In fact, at biopsy it is sometimes difficult to know whether the tumor should be classified as an adenoma or an adenocarcinoma. Of course, in either case, all the tumor should be destroyed if possible.

The case which I shall report belongs to the pseudopapillary type. A tumor of this type is microscopically almost identical with adenocarcinoma of the large bowel. It is composed of nonciliated columnar cells arranged in papilla-like formations. The stroma is composed of more or less abundant, dense connective tissue. Ringertz said of this type:

> The papillary formations in the adenocarcinomas are often constructed through extension and confluence of the glandular tubules, but may also arise through the crypt-like proliferation of the surface epithelium. The structure should therefore be called pseudo-papillary.

The symptoms of intranasal adenocarcinoma are not different from those of many other tumors in this region. The common symptoms are blocked nasal passage, blood-streaked discharge and, in some cases, severe nosebleed and facial deformities. There is seldom severe pain. Removal of parts of the tumor, even for biopsy, produces severe hemorrhage. It should be noted that this tumor is also subject to necrosis and disintegration, especially in the lower portion.

Ringertz stated that he had been unable to find any published report of metastasis in cases of this tumor and expressed the belief that the intranasal adenocarcinomas have little tendency to metastasize.

REPORT OF A CASE

A girl aged 12 years was admitted to the Riley Hospital on March 3, 1941. The chief complaint was frequent and repeated nosebleed, with some spreading of the base of the nose on the left side. Daily bleeding had been noticed for five weeks before the child was taken to the family physician. He found a growth in the left side of the nose which he diagnosed as a polyp. She was referred to the hospital, where the diagnosis on admission was also that of polyp.

A small piece of the tip of the tumor was removed on March 19. The laboratory report was that a blood clot with a small amount of granulation tissue was present. It should be noted that there was profuse bleeding at the taking of each biopsy specimen, much in excess of what might be expected in the case of a polyp.

A larger specimen, 1.5 cm. long, was removed on March 25 from the tip of the tumor, which was necrotic. The patient lost several ounces of blood at the time. Again, the laboratory reported a pyogenic granuloma, with no evidence of malignancy.

On April 23 the patient was given avertin with amylene hydrate, and an external ethmoid incision was made in order that the tumor might be approached from above. Profuse bleeding was encountered each time the tumor was touched, but, in spite of the blood, practically all the tumor was removed and the raw surfaces in the nose were cauterized by electrocoagulation. The patient was given 500 cc. of blood immediately after the operation.

The tumor consisted of a friable mass, which practically filled an ounce (30 cc.) medicine glass. It filled the anterior ethmoid cells and involved the nasoantral wall. The pathologist's report was papillary adenocarcinoma grade 2.

The patient received 500 r of radiation before the operation and 1,600 r afterward.

On June 4 a Caldwell-Luc operation was done to make sure there was no cancer tissue in the antrum and to remove any suspicious fragments of tissue. The laboratory, however, reported no evidence of malignancy in the specimens removed at this time.

During her stay in the hospital the patient had had a chronic discharge from the right ear. Pain began in this ear on March 29. Postauricular tenderness and swelling later appeared, and it was necessary to do a simple mastoidectomy on that side. Recovery was uneventful.

Fig. 1.—Photographs of the patient, showing (*A*) the spreading of the nose, due to a large intranasal tumor, and (*B*) her appearance one and a half years after operation.

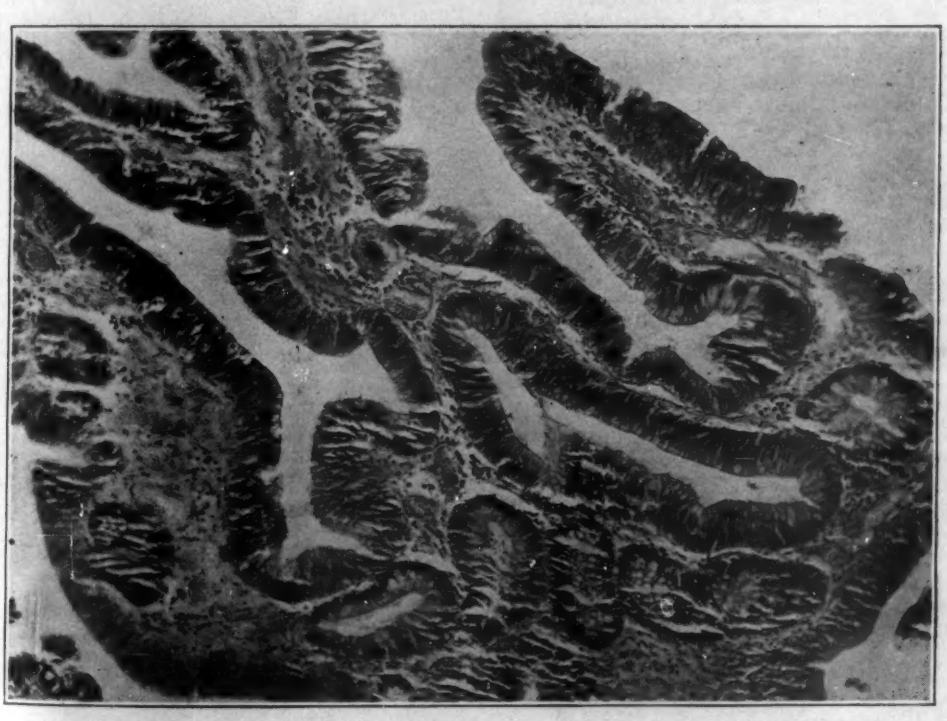


Fig. 2.—Photomicrograph, showing the large pseudopapillary formations, with a single layer of nonciliated columnar cells, the growth resembling that of cancer of the colon.

This patient has been watched closely since her dismissal from the hospital, but no tissue which even looks suspicious has yet been seen. The patient is gaining weight and is in normal health at present.

COMMENT

This case is not reported as an instance of cure, even though there is no indication of a recurrence after nearly two years. The rarity of the tumor in a child of this age makes the case seem worth reporting.

I should like, also, to comment on the method of approach in the operation in this case. I believe that in many cases of such a tumor the area of the growth can be thoroughly cleaned without resulting extensive deformity by an external ethmoidectomy, a Caldwell-Luc operation, and an intranasal operation combined. The first procedure should be largely a matter of removal of the bulk of the tumor. At a second operation the base of the tumor may be more successfully cauterized, because of better hemostasis.

Ringertz reported 10 cases of adenocarcinoma of the nasal cavity, in none of which the patient was under 20 years of age. The average age in his series was 54.1 years. In 8 of the 10 cases he reported the patient died. The 2 living patients had not been followed longer than one and a half years. The tumor is only slightly sensitive to radiation, and the only cure that can be depended on is complete removal of the growth, preferably by electrocoagulation.

Such cases as the one reported here should serve as a reminder that many such tumors are not beyond help. One must remember that when the tumor can be entirely removed and when there has not been any metastasis the patient can be cured. This case is offered to illustrate a method of removal of all the tumor without mutilation of the patient.

CONCLUSIONS

A case of an unusual type of adenocarcinoma is reported, in which the result of operation was probably favorable.

So far as I am able to determine, the patient is the youngest on record with this type of carcinoma.

A method of approach is described which can be used to advantage in operating on many tumors in the anterior portion of the nasal cavity.

303 Hume Mansur Building.

LESIONS OF THE MOUTH IN MYELOID LEUKEMIA

HENRY H. BEINFIELD, M.D., BROOKLYN

This case is presented because of the comparative rarity of the condition and to show the necessity of a proper differential diagnosis.

REPORT OF A CASE

History.—An Italian girl aged 17 was admitted to the surgical service of the Coney Island Hospital on Nov. 17, 1941, with a diagnosis of peritonsillar abscess. Cursory inspection of the mouth and pharynx appeared to confirm such an opinion, but after careful examination a diagnosis of leukemia was made. Such a mistake in diagnosis has not been uncommon—so much so that a leukemic infiltration has often been incised for a peritonsillar abscess.

The chief complaint was pronounced swelling of the left side of the face and neck for one week, with increasing inability to talk and swallow. The family and the previous personal history were without significance. Her present complaint started about one month before with symptoms referable to the upper respiratory tract. At this time the left side of the upper lip became slightly swollen and reddened. After a few days these symptoms subsided. About one week prior to admission, the left side of the face and neck began to swell. This swelling gradually increased and was accompanied by difficulty in swallowing and speech. The patient consulted a dentist, who ruled out the teeth as a cause of her complaint. Her private physician felt that it was due to an acute infection of the throat and prescribed gargles and the like. For the week prior to admission the patient had been in bed, and during this time she had had a continuous left temporal headache. Her temperature ranged between 100 and 101 F. The swelling of the face had subsided somewhat, but had returned again within the two days preceding admission.

Physical Examination.—The patient was fairly well developed but poorly nourished. She was very pale and appeared toxic and acutely ill. The left side of the face and neck were greatly swollen. This swelling was more pronounced over the area between the cheek and the ear, so that the entire face was distorted. There was moderate tenderness over this swelling, but no redness or localized heat. The cervical, postauricular, submaxillary, submental and suboccipital glands on the left side were slightly enlarged, firm and moderately fixed. The cervical glands were tender, but the others were not. There was a diffuse herpetic eruption on both lips. The gums were severely swollen and congested. There was a slight amount of trismus, and the tongue was covered with a light whitish coating. No distinctive odor from the mouth was perceptible. The floor of the mouth was apparently normal, but the left side of the pharynx in the region about the tonsil was slightly swollen and edematous. There was a punched-out, deep ulceration, about the size of a quarter, filled with a soft grayish necrotic tissue, which was somewhat adherent and bled slightly on manipulation. This ulcer involved only the left side of the hard palate posteriorly, around the molar teeth, extending from the margin of the gum to about 1 cm. short of the midline of the hard palate on the same side. The ulceration also extended to the corresponding area on the buccogingival side, with exposure of the roots of the first and second upper molars, together with the portion of the maxilla in which these teeth were set. Probing of the bare bone revealed no fistulous tracts. The larynx could not be visualized. The nose, lungs and heart were normal. The liver and spleen were not palpable. The report of dental consultation indicated that the condition might be severe Vincent's infection, possibly progressing into noma of the palate.

The blood count on admission was 68 per cent hemoglobin, 3,900,000 red cells and 47,000 white cells, with 5 per cent nonsegmented polymorphonuclears, 10 per cent segmented forms, 11 per cent lymphocytes, 15 per cent neutrophils and 59 per cent metamyelocytes.

Impression.—The clinical diagnosis was: (1) cellulitis of the left side of the face; (2) lymphadenitis of the neck; (3) suppurative parotitis; (4) necrosis of the alveolar process of the left side of the maxilla, possibly osteomyelitis; (5) Vincent's angina; (6) malignant condition of the maxilla; (7) a possible blood dyscrasia, and (8) septicemia.

Course of Illness and Further Studies.—The temperature on admission was 103 F. After receding to 101 F., it returned on the following day to 103 F., and thereafter fluctuated

Read at a meeting of the section on Laryngology, Rhinology and Otology of the Medical Society of the County of Kings, Brooklyn, Jan. 14, 1942.

between 99 and 101 F. until the time of discharge, on December 30, when the patient signed a release.

On November 19 a sternal puncture was done; biopsy of the material showed 37 per cent myeloblasts, 34 per cent myelocytes, 10 per cent metamelocytes, 4 per cent polymorphonuclear leukocytes, 2 per cent megaloblasts, 1 per cent erythroblasts, 12 per cent normoblasts and no megakaryocytes. Studies of the bone marrow revealed a notable increase of immature cells of the granulocyte series. There was a marked increase in the myeloblasts and myelocytes, with no increase of polymorphonuclears. The diagnosis was myeloblastic leukemia.

Subsequent blood counts showed a drop in the white cell count to 11,000, with a similar diminution in the polymorphonuclear cell count and an increase in lymphocytes. The hemoglobin dropped to 56 per cent and has since recovered to 65 per cent. The urine was normal except for a faint trace of albumin. A smear from the ulcerated area in the mouth showed many spirilla and fusiform bacilli. Culture of material taken from the same area showed Streptococcus viridans and a bacillus of the Friedländer group. A blood culture was sterile, and the Wassermann reaction of the blood was negative. The urea nitrogen measured 9.6 mg. and the dextrose 101 mg. per hundred cubic centimeters. The peroxidase stain of a blood smear revealed 90 per cent positive cells and 10 per cent negative cells. Roentgenograms of the left side of the maxilla, taken on November 17 and again on December 8, showed no periostitis or destruction of bone. A biopsy specimen taken from the involved area on November 28 was reported as pyogenic granuloma. On November 22 the edge of the spleen was felt at the level of the costal margin, and since then it has extended below.

Treatment.—General oral hygiene was instituted. Sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole), 15 grains [0.975 Gm.] every four hours, was given for about a week; the highest concentration in the blood reached 4.1 mg. per hundred cubic centimeters. One blood transfusion was given. No other therapy was used.

CONCLUSION

An oral lesion such as that described in this case may be the systemic manifestation of any of several diseases, such as myeloid leukemia, syphilis, tuberculosis, agranulocytosis, Vincent's angina, metal toxemia, infectious mononucleosis, neoplasm or osteomyelitis of the superior maxilla. A punched-out, sloughing ulceration with a blood count as previously described created the impression that the case was one of myeloid leukemia, a diagnosis which was confirmed by a sternal puncture. The absence of angry, red edges around the ulcer and the negative serologic reactions ruled out syphilis. The depth of the ulcer and the absence of any systemic evidence eliminated tuberculosis. Naturally, the blood picture immediately dissipated the possibilities of agranulocytosis and infectious mononucleosis. Although the dental department made a diagnosis of Vincent's angina on the basis of the presence of the organisms, I did not hold such an opinion, in view of the extreme depth of the ulcer, the pronounced surrounding induration and the comparative lack of pain, as well as because Vincent's spirilla and fusiform bacilli are frequently found in the ulcerations associated with myelogenous leukemia. Osteomyelitis of the maxilla was eliminated by negative roentgenographic evidence. The presence of a neoplasm was completely negated by biopsy. Metal poisoning might readily simulate an oral lesion of this kind. A negative history of exposure to metals removed such a possibility.

Like the ophthalmologist in his field, the otorhinolaryngologist can diagnose certain systemic conditions which involve lesions of the ear, nose and throat. Some such conditions, in addition to those already mentioned, are diabetes, the exanthems, avitaminosis, hysteria, Addison's disease and the anemias.

This case illustrates the fact that otolaryngology is not a closely defined specialty. It is, rather, an integral part of medicine, and the otolaryngologist should always be on the alert to link his findings with some general systemic disorder. Finally, he should always remember that many an obscure disorder is hidden behind a screen of leukemia, the oral lesions being merely a local manifestation of a systemic disease.

760 Eastern Parkway.

Progress in Otolaryngology

A Summary of the American and British Bibliographic Material Available in the Field of Otolaryngology

TONSILS AND ADENOIDS

J. DUDLEY SINGLETON, M.D.
DALLAS, TEXAS

In reviewing the American and British literature on tonsils and adenoids for the year beginning Oct. 1, 1941 one finds a notable decrease in the number of articles published on this subject. However, in spite of the scarcity of material, several interesting and worth while articles have appeared. Several papers have been published on the relation of tonsillectomy to poliomyelitis. Postoperative complications and infections attracted considerable attention, and benign tumors of the tonsils have been discussed by a few authors. The adenoid was largely neglected; it was mentioned only casually in connection with the tonsils and the nasopharynx.

TONSILS: GENERAL CONSIDERATIONS

Unger [1] calls attention to the fact that analyses have been made by many students in recent years which have caused considerable doubt concerning the value of tonsillectomy in alleviating or curing such conditions as heart disease, rheumatism, nephritis, pulmonary disease and gastrointestinal trouble. He then states:

> . . . It behooves the medical profession to reexamine the evidence in its possession on the rôle that the tonsil plays as a disease dispersing factor and to reappraise the value of tonsillectomy, with the purpose in mind of confirming it as the procedure of choice, of modifying it, or of discarding it, as may best serve the needs of the patient.

Unger believes that most of the good results that follow the removal of tonsils and adenoids in children accrue from the removal of the adenoid. He does not believe that the tonsil plays an important role as a focus of infection and gives several references in support of this view. He points out that in the removal of tonsils in adults the good results obtained are local, and consist primarily in the relief and prevention of sore throat and peritonsillar abscess. In his discussion he says:

> It [the tonsil] is simply a lymph gland, like other lymph glands. Like them it becomes involved when infection develops in the region which it drains. The special feature which distinguishes the tonsil from other lymph glands is the presence of crypts. But is it necessary to remove the whole tonsil and destroy its barrier effects because one or more crypts are pockets of pus?

Unger favors free incision and drainage of the crypts by cryptotomy. The technic of cryptotomy is explained, and a knife for performing the operation is illustrated and described.

Emenhiser [2] states that the tonsils should be removed when they are so large as to obstruct respiration or when they harbor infection. Chronic hypertrophy

1. Unger, M.: Tonsillectomy, Adenoidectomy and Cryptotomy, M. Rec. **155**:119-122 (Feb. 18) 1942.
2. Emenhiser, L. K.: Too Many Tonsillectomies! Mil. Surgeon **91**:182-185 (Aug.) 1942.

of the tonsil, persistent hyperemia of the pillars, enlargement of the cervical lymph nodes, frequent severely sore throat, a previous attack of quinsy and suppurative cervical adenitis are all indications of infection of the tonsils. He does not believe that the consistency of the tonsil or the presence of a yellowish secretion in the crypts is a reliable guide in making a diagnosis of infected tonsil. The author states that he has recently written to many internists and pediatricians asking their opinion on the value of tonsillectomy in the treatment of disease that might be due to focal infection. Almost without exception they replied that the results of tonsillectomy were of doubtful, little or no benefit, either prophylactically or therapeutically, except rarely in cases of such conditions as rheumatic arthritis, nephritis, chorea, heart disease, rheumatic fever, rheumatic carditis, tuberculosis, common colds, impairment of hearing, chronic purulent otitis media, muscular or growing pains, nasal and pulmonary allergy, headaches of unknown origin, hypertension, asthma, sinusitis, the so-called streptococcic sore throat, gastrointestinal disease, enuresis and halitosis. He concludes that the preservation of the human race or the survival of the fittest does not depend on tonsillectomies.

Coates and Gordon,[3] in an able discussion on the indications for tonsillectomy, review briefly some of the fundamental principles in the diagnosis and treatment of disease. They give Barnhill's definition of a focus of infection and call attention to the fact that it has been estimated that 75 per cent of all primary foci of infection occur in the mucous membranes of the head, particularly about the roots of the teeth and in the crypts of the tonsils and adenoids. Focal infection in parts of the body remote from the head may be primary or secondary. Active foci of the appendix, kidney, gallbladder and intestinal tract are often secondary to a primary focus in the upper respiratory or upper digestive tract. Primary infection may extend to secondary areas by (1) direct extension, (2) the lymphatic vessels or (3) the blood stream. The last is the most frequent pathway of extension.

The authors review in considerable detail the gross clinical anatomy of the tonsils and adenoids and to a less extent their histology and histopathology. In discussing the lymphatics of the tonsils, they call attention to the fact that the tonsils have efferent, but no afferent, lymphatic vessels.

Indications for tonsillectomy are considered under three classes, as follows: (1) conditions within the tonsils; (2) conditions adjacent to the tonsils, and (3) conditions remote from the tonsils. Conditions within the tonsils indicating tonsillectomy are: (1) repeated attacks of tonsillitis; (2) abscess within the tonsils; (3) epidemic streptococcic sore throat; (4) tuberculous tonsils; (5) diphtheritic tonsils; (6) hypertrophied tonsils; (7) Vincent's infection of the tonsils, and (8) halitosis due to infection of the tonsils.

Conditions adjacent to the tonsils indicating tonsillectomy are: (1) acute catarrhal and suppurative otitis media; (2) chronic suppurative otitis media; (3) middle ear deafness; (4) labyrinthitis and inner ear deafness; (5) sinusitis; (6) thyroid disease; (7) laryngitis; (8) pharyngitis; (9) cervical adenitis; (10) peritonsillar abscess; (11) acute phlebitis of cervical veins, and (12) ocular diseases. Tonsillectomy should be performed prior to submucous resection or any surgical procedure on the sinuses, abdomen, lung or eye.

In discussion of conditions remote from the tonsils that indicate tonsillectomy, a long list of acute and chronic processes traceable to a primary focus of infection is cited. In the management of such infections the closest cooperation of the

3. Coates, G. M., and Gordon, W.: Indications for Tonsillectomy, Pennsylvania M. J. 45:218-225 (Dec.) 1941.

internist, the pediatrician or the general practitioner is advised. The authors deplore hastily considered data, and they scorn injudicious tonsillectomies.

The article is concluded in part as follows:

> Each patient must be individually studied, surveyed and observed. The closest cooperation between the otolaryngologist and the internist and anyone connected with the given case is of the utmost importance. Tonsillectomy should be performed only when indications warrant it. Tonsillectomy is very important and when performed properly and completely at the proper time, in conjunction with the broad principles of management, will produce the desired effect. The diagnosis of tonsillar sepsis must be exact; the surgical removal of this focus must be precise and skillfully performed.

Shambaugh,[4] in a lengthy and practical discussion of the tonsils and adenoids, states that they should be removed when there is evidence that they are the seat of chronic infection which is producing, or threatens to produce, bodily injury greater than the dangers, disability, discomfort and inconvenience of removal. He then gives a list of ten indications for the removal of the tonsils and adenoids, calls attention to several local signs of chronic tonsillitis and discusses briefly the age limit for tonsillectomy. He continues his discussion in detail under several headings, as follows: (1) technic of examination of tonsils and adenoids; (2) preoperative measures; (3) technic of tonsillectomy with the patient under general anesthesia, with especial attention to (*a*) position of the head and the type of anesthesia, (*b*) removal of the adenoids, (*c*) removal of the tonsils, (*d*) postoperative care and (*e*) postoperative complications; (4) technic of tonsillectomy with local anesthesia, with particular attention to (*a*) position of the patient and the type of anesthesia, (*b*) removal of the tonsils and (*c*) postoperative care, and (5) recurrence of tonsils and adenoids. This is an article well worth reading.

McAuliffe and Leask [5] have devised and developed an instrument designed to apply direct mass suction to the tonsil and, at the same time, to irrigate the tonsillar crypts. The instrument is illustrated and described.

The authors state that cryptal plugs are conducive to tonsillar infection by blocking the passage through which the tonsil normally rids itself of cells and bacteria. The plugs provide an environment, with varying degrees of oxygen tension and other factors, which is ideal for the growth and propagation of both aerobic and anaerobic organisms, and favors the retention and absorption of toxic metabolic products. Many of the organisms are capable of causing necrosis in the deep epithelial lining of the crypt, with ensuing tonsillar infection. Aeration and circulation are interfered with, with resultant pressure on adjacent tissues, a condition which may in turn produce congestion, inflammation and thrombosis.

The authors have found wet suction at 15 pounds' (6.8 Kg.) pressure to be an effective means of ridding the tonsillar crypts of accumulated organisms, cells and debris, congestion and inflammation being thereby relieved; by making cultures of the sediment from the centrifuged irrigation fluids, the bacterial flora of an infected tonsil can be determined accurately before tonsillectomy, and cytologic examination of these sediments is helpful in differentiating between hypertrophy of the tonsil due to infection and that due to normal endocrine function.

Gerrie and Mackenzie [6] report on the oral administration of pentobarbital sodium before operation to over 600 children, ranging in age from 9 months to 12

4. Shambaugh, G. E., Jr.: Technique of Tonsillectomy and Adenoidectomy, S. Clin. North America **22**:237-252 (Feb.) 1942.

5. McAuliffe, G. W., and Leask, M.: Laboratory Analysis of the Contents of Tonsil Crypts as Obtained by the Wet Suction Technic, Arch. Otolaryng. **34**:758-770 (Oct.) 1941.

6. Gerrie, J., and Mackenzie, J. R.: Basal Narcosis for Tonsil Operations on Children, Lancet **1**:759-760 (June 27) 1942.

years. The purpose of the basal narcotic is to protect the patients as far as possible from the mental disturbances which are inherent in the operation.

Briefly, the technic is as follows: City patients arrive at the hospital at 8 a. m., while children from the country are admitted on the previous afternoon. The patient is undressed, weighed and put to bed. No hypodermic is given unless dissection is contemplated, when it is given immediately before the basal narcotic. At 9 a. m. each patient receives by mouth a dose of pentobarbital sodium, equivalent to ¾ grain (0.048 Gm.) per stone (6.4 Kg.) of body weight. At 9:15 a. m. the ward is darkened, and the side of each cot is let down in preparation for the noiseless removal of the patients to the anesthetic room. Patients over 1 year old who arrive in the afternoon receive 1 grain (0.065 Gm.) of pentobarbital sodium in the evening and in the morning ½ grain (0.032 Gm.) per stone of body weight, plus ½ grain for any fraction of a stone. At 10:15 a. m. the first patient is carried to the darkened theater and is given ethyl chloride on a perhalation mask. As soon as stertorous respiration appears a mouth gag is inserted. The tonsils are enucleated with the Layton hemostatic guillotine and the adenoids curetted if necessary. Blood is wiped out of the mouth, and the patient is carried back to bed and the side of the cot put up. No bad results have been observed.

[Comment: I do not approve of the use of any anesthetic or operative technic that does not permit complete control of all bleeding before the patient leaves the operating room.]

Kaiser [7] presents a follow-up study of 4,400 children over a ten year period, half of whom had their tonsils and adenoids removed and the other half of whom did not have surgical treatment. Initial examination was made and observation on these children begun when they were between the ages of 4 and 7 years, and the observations were continued into their high school period. The study showed that approximately 38 per cent of the 4,400 children were subject to repeated attacks of tonsillitis (at least two febrile attacks a year) during the first seven years of life. Tonsillitis or sore throat occurred less frequently in the patients whose tonsils had been removed. [The reviewer wonders why tonsillitis and sore throat are used here synonymously.] Furthermore, the common cold (four colds or more a year) occurred with about equal frequency in the two groups under observation. During the three year period following the tonsillectomy and adenoidectomy, the incidence of purulent otitis media was considerably lower in the group of children who were operated on than in the group who were not operated on. It is Kaiser's impression that removal of the adenoids was responsible for this advantage, for during the next seven years the children not operated on fared as well, or even a little better, than those who were operated on.

Kaiser further brought out the fact that during the ten year period following operation the incidence of cervical adenitis in the children who were operated on was 7 per cent as compared with an incidence of 14 per cent for the children who were not operated on. He feels that children with acute sinusitis, nasal allergy and allergic involvement of the sinuses are not favorably influenced by operations on the tonsils and adenoids. He is of the same opinion with regard to laryngitis.

It was noted that pulmonary infections occurred more frequently in children during the first five years of life than in the next ten years. There were, however, more pulmonary infections in the group operated on over the ten year period than in the control group. According to the author, either the absence of the tonsils

7. Kaiser, A. D.: Effect of Tonsillectomy on Respiratory Infections in Children, Bull. New York Acad. Med. **18**:338-346 (May) 1942.

increased the chances of development of bronchitis and pneumonia or the tonsillectomized group represented the more susceptible persons and the operation was not responsible for the increased incidence of pulmonary infection. The author believes that there is suggestive evidence that the absence of the tonsils may encourage pulmonary involvement because the soil in which bacteria might develop has been removed in the oral cavity. Kaiser concludes that the removal of the tonsils and adenoids when indicated does reduce the incidence of infections of the upper respiratory tract, even though their removal does not tend toward a lower incidence of pulmonary infections.

Zerffi,[8] in discussing the effects of tonsillectomy on the singing voice, states that in earlier days much damage was done to singers' throats by the removal of tonsils. However, with the improvement in technic, the danger has been so greatly reduced that it is unusual to find a case in which the throat has suffered severe damage. He discusses briefly the function of the soft palate in voice production and credits Makuen with the statement that "the soft palate has two important functions in voice production: (1) its valvular function in influencing vocal resonance and (2) its power of tilting the thyroid cartilages, thereby stretching the vocal cords and in this way influencing the quality and pitch of the voice."

In performing a tonsillectomy one must avoid damage to the soft palate and to the palatopharyngeus and stylopharyngeus muscles. These muscles are attached to the rear of the thyroid cartilage, and their action tilts the cartilage and helps to tense the vocal cords. After tonsillectomy there should be complete cessation from singing from four to six weeks. A singer should be advised to resume singing with very light exercises and frequent intervals of rest. The author stresses the dangers that may follow attempts to make full use of the voice before the throat is thoroughly healed.

Maxey, Pharr and Smith [9] have reviewed and analyzed twelve diphtheria carrier surveys made by the department of epidemiology of the Johns Hopkins School of Hygiene and Public Health from 1921 to 1940. The studies involved 24,243 white and Negro children in the grade schools of Baltimore. The children were students in two age groups, the first from 5 to 9 years and the second from 10 to 14 years. As a result of their study, the authors found (1) a higher carrier rate among the white children, (2) a higher carrier rate among children in the younger age group and (3) a higher carrier rate in children whose tonsils and adenoids had not been removed.

Ashley [10] made a study on 1,524 school children in a community under average normal conditions of American life for the purpose of securing data on school attendance and gain in weight for tonsillectomized and for nontonsillectomized children. Of the total number, 602 children had previously had their tonsils removed and 922 had not had their tonsils removed. The tonsillectomized group lost slightly less time from school than did the nontonsillectomized group, but the difference was so slight as to be insignificant. When the gains in weight were tabulated, it was found that the total gain was greater for the tonsillectomized group, and that the total gain in weight in the first year in school for the tonsillectomized group was almost twice that for the nontonsillectomized group.

8. Zerffi, W. A. C.: Tonsillectomy and Its Effect on the Singing Voice, Arch. Otolaryng. **35**:915-917 (June) 1942.

9. Maxey, K. F.; Pharr, J. J., and Smith, M. R.: Diphtheria in Baltimore: Tonsillectomies as Related to the Diphtheria Carrier Rates, Am. J. Hyg. **35**:42-46 (Jan.) 1942.

10. Ashley, C. W.: A Note on the Tonsil Question, Pennsylvania M. J. **45**:594-595 (March) 1942.

Ross [11] reports an unusual case in which a third molar tooth was removed from the region of the lower pole of the left tonsil during a tonsillectomy. The history indicates that the tooth had become embedded in this region nineteen years previously, after the extraction of seven teeth on the left side. One week after this extraction quinsy developed, but the patient had no further trouble, except for occasional sore throat, until a few months prior to the tonsillectomy and the removal of the embedded tooth.

Monteiro [12] calls attention to one of his previous publications on the treatment of acute lacunar tonsillitis by the injection of a bismuth compound and reviews some of the recent work of various authors on this subject. Monteiro and other investigators report rapid relief of pain, reduction of temperature and elimination of the follicular exudate following the injection of bismuth compounds. The method is most effective in the treatment of streptococcic infection. The dose varies with the age of the patient and the solubility of the bismuth preparation used.

Brakeley and Shaul [13] discuss in detail the importance of thorough physical examinations prior to tonsillectomy. They cite Hunt to the effect that the taking of the bleeding and coagulation times are of little value in determining which patients are most likely to have hemorrhage. They believe the elimination of hemophilia and other familial dyscrasias by means of a careful history is much more important in the prevention of post-tonsillectomy hemorrhage than determination of the coagulation and bleeding times.

Shambaugh [14] describes and illustrates a special tonsil knife and adenotome for the removal of tonsils and adenoids when the patient is under general anesthesia, with the head in the hyperextended position. The knife has a straight blade at one end; at the other there is a curved blade with a blunt point and both edges sharp. The adenotome is similar to the regular La Force instrument except that the curve is reversed.

RELATION OF TONSILLECTOMY TO POLIOMYELITIS

Aycock,[15] in a comprehensive study on the relation of tonsillectomy and poliomyelitis, states:

> The present paper is a review and analysis of data available in relation to poliomyelitis following tonsillectomy, a selectivity which is important as indicative of the *modus operandi* of autarcesis—an understanding of which might offer hope of bringing under control the one serious consequence of infection with the virus—and which already provides a means of preventing numbers of cases of a distressing and highly fatal form of the disease.

The thoroughness of this study is indicated by the vast amount of data collected and analyzed from various sources, including hospitals, city and state health departments, numerous personal communications and fifty-two articles in the literature. The study is briefly summarized in the author's conclusions as follows:

> The results of the present study are in keeping with other epidemiologic indications that some added circumstance determines the form of the disease which develops upon exposure to the virus of poliomyelitis and that this circumstance resides not so much in parasitic factors or in environmental conditions affecting exposure but in factors of autarcesis in the host.

11. Ross, A. M.: Misplaced Wisdom Tooth Extracted During Tonsillectomy, J. Laryng. & Otol. **56**:437-438 (Dec.) 1941.

12. Monteiro, A.: Modern Treatment of Acute Tonsillitis by Injection of Bismuth Compounds, Arch. Otolaryng. **34**:719-722 (Oct.) 1941.

13. Brakeley, E., and Shaul, J. F.: Pediatric Aspects of the Tonsil and Adenoid Problem, Arch. Pediat. **59**:347-355 (June) 1942.

14. Shambaugh, G. E., Jr.: Tonsil Knife and Adenotome for Use with Head in Hyperextended Position, Arch. Otolaryng. **35**:934 (June) 1942.

15. Aycock, W. L.: Tonsillectomy and Poliomyelitis, Medicine **21**:65-94 (Feb.) 1942.

It is clear from our tabulations that there is a casual relationship between the removal of tonsils and the onset of bulbar poliomyelitis within the time interval corresponding to the incubation period of the disease.

The figures collected do not lend themselves to analysis as to whether or not the absence of tonsils predisposes to clinical poliomyelitis, with one exception. In this instance, cases of poliomyelitis gave a history of removal of tonsils with greater frequency than controls in the same area. Our tabulations do show, however, that the relative frequency of the occurrence of the bulbar, as compared with the spinal, form of poliomyelitis is greater at all ages in persons giving a history of previous tonsillectomy.

Because of the numerical considerations, the hazard of bulbar rather than spinal poliomyelitis in tonsillectomized individuals hardly constitutes in itself a contraindication to the operation. Although the risk should be recognized, the decision for the operation should rather be based upon the indications in the individual case.

The selectivity seen in the occurrence of bulbar poliomyelitis following tonsillectomy and its relatively more frequent occurrence in tonsillectomized individuals comprise a demonstration that the nasopharyngeal mucosa may be the locus of at least one added circumstance which determines the outcome of exposure to the virus, and is important not only as indicative of the *modus operandi* of autarcesis, but as providing a means of prevention. Since this operation is practically always elective as to time, changing the season when it is done, so as not to coincide with the season of poliomyelitis prevalence, would eliminate numbers of cases of the distressing and highly fatal bulbar form of the disease.

Francis, Krill, Toomey and Mack [16] report the occurrence of 5 cases of the bulbar type of poliomyelitis in a family of 6 children following general tonsillectomy. The youngest child, aged 2½ years, was not operated on and did not contract the disease. In an effort to determine the source of the infection the authors made extensive epidemiologic studies combining the etiologic and the laboratory approach. The activities of the family were reviewed in detail and presented in chronologic order. Virus studies were made on the stools of 54 persons, and in collection of the stools emphasis was placed on the children's associates. Virus was found in the stools of the youngest child who was not operated on, in the stools of 6 cousins with whom the children had visited and lived in intimate contact prior to their operation and in the stools of 3 children of a family who were close playmates. The authors are of the opinion that tonsillectomy performed on an infected subject is the provocative factor in precipitating the bulbar type of poliomyelitis in a person who otherwise would probably escape with inapparent infection.

Seydell,[17] in an extensive study on the relation of tonsillectomy to poliomyelitis, reviewed the available literature and made an analysis of statistics which he obtained from public health authorities, hospitals and a survey of a severe epidemic of poliomyelitis in Kansas in 1940. The investigation was made because of the appearance of a number of articles in the literature in the last twelve years reporting the occurrence of poliomyelitis, especially the bulbar type, following the removal of tonsils during epidemics of the disease.

As a result of his studies, Seydell states he is unable to show a definite relation of tonsillectomy to poliomyelitis. He concludes his article in part as follows:

It is impossible for me to draw any definite conclusions relative to the relation of tonsillectomy to poliomyelitis from the data that I have been able to gather from the various articles that have appeared in the literature on this subject or from my own survey.

16. Francis, T., Jr.; Krill, C. E.; Toomey, J. A., and Mack, W. N.: Poliomyelitis Following Tonsillectomy in Five Members of a Family on Epidemiologic Study, J. A. M. A. **119**:1392-1396 (Aug. 22) 1942.

17. Seydell, E. M.: Relation of Tonsillectomy to Poliomyelitis, Arch. Otolaryng. **35**:91-106 (Jan.) 1942.

COMPLICATIONS

Iglauer [18] reports in detail a case of septic thrombophlebitis of the cervical veins following an apparent attack of tonsillitis. The outcome of this rather infrequent complication depends in large measure on its early recognition and the employment of proper surgical and medical treatment.

The author favors the use of local anesthesia for the operative work. The parapharyngeal space should be opened and drained through an external incision, and the internal jugular, the common facial and the posterior facial vein should be exposed and inspected. If the veins are thrombosed or collapsed, they should be ligated and removed and the exploration carried as near the tonsil bed as possible. It may be necessary to sever the posterior belly of the digastric muscle and the stylohyoid muscle in order to reach the primary source of infection. The tonsil on the affected side should be removed, preferably at a subsequent operation.

Hara and Courville [19] report in detail 2 cases of intracranial complications of tonsillar origin and review the available literature on the subject since 1900, which includes fifty-nine references. In a survey of 25,000 autopsies performed in the Los Angeles General Hospital, they found 1 case of an intracranial complication of tonsillar origin. As a result of their survey, they noted that most such intracranial complications occur in the form of sinus thrombosis or septic meningitis. The infection reaches the intracranial space by one or more of four possible routes: (1) by way of the perineural sheaths of the extracranial nerves; (2) by way of tissue spaces; (3) by arterial channels, and (4) by venous channels. Thrombosis of the venous sinuses seems to follow a more or less direct pathway from the jugular vein into the lateral sinus, or it may spread from the pharyngeal, pterygoid and associated plexuses into the cavernous sinus. The authors are of the opinion that the avoidance of surgical trauma in the presence of acute infections of the tonsils will probably prevent many intracranial complications from this source.

Gorrell,[20] in discussing the control of delayed hemorrhage following tonsillectomy, writes:

> The necrotic tissue cannot be grasped in the usual manner with a hemostat; sutures tend to cut through; local applications are of temporary effectiveness only; injections of the various sera are ineffective. Bleeding usually occurs in patients with infected tissue, so that the more manipulation is carried out, the more chance there is of spreading infection both locally and in the blood stream.

He then reports 2 cases of post-tonsillectomy hemorrhage in which blood transfusions were given, with complete cessation of all bleeding, a result which he attributes to the transfusions. In 1 case local measures were tried after the area was anesthesized with powdered cocaine and solution of nupercaine. He also reports a case in which another physician operated and a sudden fatal spontaneous hemorrhage occurred two weeks after tonsillectomy, due apparently to a deep cervical abscess with erosion of one of the large arteries.

[Comment: In attempting to control late post-tonsillectomy hemorrhages, I never use cocaine in the throat in any form because of the danger from rapid absorption. In cases of severe hemorrhage blood transfusions are certainly indi-

18. Iglauer, S.: Septic Thrombophlebitis of the Neck, Arch. Otolaryng. **36**:381-384 (Sept.) 1942.

19. Hara, H. J., and Courville, C. B.: Intracranial Complications of Tonsillar Origin, Arch. Otolaryng. **35**:530-571 (April) 1942.

20. Gorrell, R. T.: Treatment of Delayed Tonsillectomy Hemorrhage, Am. J. Surg. **57**: 147-150 (July) 1942.

cated and should be used. However, it has been my experience that most late hemorrhages are easily controlled by thorough, gentle removal of the clot, followed by firm application to the bleeding area of some such agent as a solution of tannic and gallic acid.]

Cunningham [21] reports on a study of 7 adults on whom tonsillectomies were performed. All bleeding was completely controlled before the patients left the operating room. After a lapse of several days sanguineous oozing occurred from one of the tonsillar fossa. After the ordinary methods to control the bleeding had failed (sedation with morphine, cleansing of the fossa and pressure) sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) powder was applied with a powder blower. Prompt hemostasis followed.

Further observations were carried out on laboratory animals. Here finely ground powder of sulfanilamide, sulfapyridine, sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole), sulfamethylthiazole (2-[paraaminobenzenesulfonamido]-4-methylthiazole) and talc were spread evenly over the bleeding surface of separate wounds; one wound was left entirely untreated. The wounds treated with sulfapyridine and sulfamethylthiazole showed an immediate tendency toward hemostasis. It was further noted that the wounds treated with any of the chemotherapeutic agents had much less edema and inflammatory reaction than the talc-covered and the untreated wounds. A 40 per cent solution of sulfapyridine was found to be equally as effective as the powder in producing hemostasis in the experimental work.

Judge [22] presents the cases of 2 young adults on whom tonsillectomy was performed with the use of general anesthesia. In each patient dense adhesions were present between the capsule and the muscle layer on one side. One of the patients had fairly profuse bleeding. Three hours after operation she complained of a painful swelling beneath the angle of the mandible of the side on which the adhesions were encountered. Examination revealed a small, tender area of crepitation. This disappeared in twenty-four hours. In the other case a tear in the muscle bed was noted. This was closed with a suture of plain catgut. Approximately two hours after the operation a swelling was noted about the eye and cheek on the corresponding side. Further examination revealed tender swellings in the neck and on the upper wall of the chest on the same side. These swellings were all crepitant. The emphysema had entirely disappeared when the patient was examined one week later.

Waddell and Birdsong [23] report a case of tularemia in a child with local lesions confined to the tonsils. On admission of the patient to the University of Virginia Hospital, the throat and tonsils were decidedly inflamed, with the tonsils practically meeting in the midline. The tonsillar crypts contained a large amount of caseous material. The cervical glands were moderately swollen and tender. After admission to the hospital the patient's illness continued to run a septic course, and repeated blood transfusions were necessary. A further history revealed that the child had helped skin a number of rabbits several days before the onset of her illness. Because of the later history, agglutination tests for tularemia were performed and gave positive results with dilutions up to 1 : 1,260.

---

21. Cunningham, B. P.: Clinical and Experimental Studies with Sulfapyridine as a Hemostatic Agent, Ann. Otol., Rhin. & Laryng. **51**:301-316 (June) 1942.

22. Judge, A. F.: Subcutaneous Emphysema Following Tonsillectomy, New York State J. Med. **42**:1359-1360 (July 15) 1942.

23. Waddell, W. W., Jr., and Birdsong, M.: Tularemia with Local Lesions Confined to the Tonsils: A Case Report, J. Pediat. **20**:368-369 (March) 1942.

BENIGN TUMORS OF THE TONSIL

Henner,[24] in the course of examinations of the throat in the Sixth Corps Area Army Induction Board, Chicago, was impressed by the frequency of papilloma of the tonsil and uvula. In a brief review of the literature, he found that Vidau, in a survey of the world's literature on papilloma of the tonsil in 1927, reported only 16 cases. In 1931 New and Childrey reported 10 cases encountered from 1917 to 1930, and in 1938 Frank recorded 3 cases seen over a period of six years. In 25,000 examinations Henner found 94 papillomas, or 1 in every 266 men examined. As a result of his observations, Henner believes that papilloma should be considered a common tumor in the tonsillar area.

[Comment: I agree with Henner that papilloma of the tonsil and uvula is by far the most common tumor in this area.]

Thomson [25] reports removal of a fibroadenolipoma of the tonsil from a man aged 55 years. The patient had first noticed the tumor about four months prior to its removal. It was slow growing but painless. The mass was oval and about the size of a walnut. It was attached by a broad base to the left tonsillar region. The mucous membrane over it was normal, and the glands in the neck were not enlarged. The tonsils had been incompletely removed before. At operation the tumor and a fair-sized tonsillar mass were removed in one piece. There had been no recurrence at the end of one year. Microscopic examination showed patches of well differentiated tissue of the salivary glands and many bundles of unstriped muscle fibers running in various directions through the bulk of the tumor, which consisted of fatty tissue.

Frank [26] reports a case of plasmocytoma of the tonsil. The patient, a man aged 40, complained of slight difficulty in swallowing for two months. Examination revealed a polypoid structure, 6 mm. in length, projecting from the right tonsil. The tumor was removed surgically and had not recurred in a period of three years.

In a review of the literature, Frank found approximately 50 cases of this type of neoplasm. He gives a comprehensive pathologic report and a thorough differentiation of this neoplasm from atypical Hodgkin's granuloma and myeloma of the plasma cell variety—the so-called "granuloma plasma cellulare."

ADENOIDS

Ridges [27] reviews the embryology and anatomy of the nasopharynx in considerable detail. He defines adenoids as a pathologic enlargement of the pharyngeal tonsil and discusses briefly its relation to progressive deafness in children. When surgical removal of the adenoid is indicated, he states that the operation should be done under direct vision with a "razor-sharp" curet large enough to engage the entire adenoid mass. After removal, all bleeding should be controlled before the patient leaves the operating room.

1719 Pacific Avenue.

24. Henner, R.: Papilloma of the Tonsil and Uvula, Arch. Otolaryng. **35**:810-811 (May) 1942.

25. Thomson, L. C.: A Fibroadenolipoma of the Tonsil, J. Laryng. & Otol. **56**:368-369 (Oct.) 1941.

26. Frank, I.: Plasmocytoma of the Tonsil, Ann. Otol., Rhin. & Laryng. **51**:22-28 (March) 1942.

27. Ridges, A. J.: The Nasopharynx, Southwestern Med. **26**:255-261 (Aug.) 1942.

Abstracts from Current Literature

Ear

OTOLOGY AND AVIATION. RALPH A. FENTON, Ann. Otol., Rhin. & Laryng. **51**:333 (June) 1942.

Applicants for flying training are rejected if there is any abnormality of the ear. The external canal must be free of infection or obstruction. Any inflammation, perforation or thin atrophic scar disqualifies, but firmly healed scars, not impeding motion, are passed. Radical mastoidectomy disqualifies, but a simple mastoidectomy with proper healing does not. A history of otitis media without recurrence in six months and without previous attacks, is not disqualifying if hearing and other tests give normal results. Repeated acute exacerbations are cause for rejection. Persistent or recurrent tinnitus for more than six months disqualifies, particularly if associated with local pathologic changes. Hearing must be 20/20 on original examination with the whispered voice. Audiometric examination is made when possible, and pilots must not show a loss of over 15 per cent on original examination. If the drum heads are retracted the eustachian tubes are examined for patency under vision. Blockage disqualifies all applicants. Except when there is a history of vestibular instability, such as train, sea or air sickness, self-balancing tests are the only vestibular examinations made. When this test shows unsteadiness, the history is suspicious, there is pronounced tremor of the eyelids and fingers, a Romberg sign or unsteady gait or the pulse rate is above 90 when the applicant is sitting, tests are made in the turning chair. The caloric test is not ordinarily used in military examinations.

Effects of flight on hearing depends on noise and vibration and the changes in air pressure. Helmets and rubber-cupped radio head sets somewhat mask the noise and vibration. Cotton or rubber ear plugs are of some help. Sound-proofing the cabin type planes eliminates much of the damage due to noise caused by exposure in open cockpits. Changes in air pressure interfere with hearing for the lower range of conversational tones. Equalization of pressure is usually readily accomplished on ascent, but during descent, if a speed of greater than 300 feet (90 meters) a minute is reached, swallowing will open the tubes with increasing difficulty, until they are held tightly closed by the increasing pressure and cannot be opened at all. Acute aero-otitis-media then develops. Repeated exposure to this hazard will lead to chronic fibrotic changes and reduction in perception of low tones. Catheterization or politzeration gives rapid relief. Patients should not be permitted to fly when they have any acute rhinopharyngeal infection. The end results of power diving and those of rapid climbing are the same, although diving produces more acute effects. Rapid ascent to above 15,000 to 20,000 feet (4.5 to 6 kilometers) may so quickly lower intratympanic pressure that the sudden shock, added to the effects of anoxia, may well contribute the deciding factor to a dangerous loss of consciousness in high flight. Air sickness may be caused by excessive stimulation of the labyrinth due to rapid maneuvering, but visual impressions and visceral disturbances usually contribute. Smoking is probably not harmful except to those with diminished tolerance to tobacco, rapid heart or high blood pressure. Alcohol, even in small amounts, releases inhibitions and may lead to recklessness. As alcohol itself produces anoxia, it must not be used before maneuvers, particularly high altitude flying, as it lowers the flyer's "ceiling." The increase in blood pressure and heart rate due to the alcohol adds to the danger of aero-otitis-media. Temporary psychic instability predisposes to circulatory upsets and thus to interference with the tympanum and labyrinth. Indiscriminate use of ephedrine and benzedrine is unwise, as these drugs produce secondary congestion. Gas embolism at high altitudes has not been considered a factor in "blacking out."

M. V. MILLER, Philadelphia.

THE ETIOLOGY OF MYRINGOTIS BULLOSA HAEMORRHAGICA. BEN H. SENTURIA and S. EDWARD SULKIN, Ann. Otol., Rhin. & Laryng. **51**:476 (June) 1942.

The authors make a preliminary report of their investigation of the cause of myringitis bullosa haemorrhagica, giving the results of their tests, which seem to prove that no relationship exists between the condition and the known types of influenza.

M. V. MILLER, Philadelphia.

BILATERAL JUGULAR LIGATION FOLLOWING BILATERAL SUPPURATIVE MASTOIDITIS. MAURICE G. EVANS, Ann. Otol., Rhin. & Laryng. **51**:615 (Sept.) 1942.

A case of recovery is reported, together with a review of the literature. From experience and investigation, the author feels that there is no firm basis for the fear of ligating both internal jugular veins, should such a necessity arise. The procedure is not necessarily fatal, nor does it follow that intracranial symptoms must occur when the venous return from the cranial cavity is materially influenced by the operation, as sufficient collateral circulation is soon established.

M. V. MILLER, Philadelphia.

MODERN ASPECTS OF THE HEARING AID PROBLEM. THOMAS H. HALSTED and FREDERICK M. GROSSMAN, New York State J. Med. **42**:1944 (Oct. 15) 1942.

The authors have been making a clinical study of various types of hearing aids for many months. Two general varieties are in use: (1) the carbon microphone, which transmits low frequencies well, and (2) the crystal microphone, which favors the high frequencies. Crystal microphones must have vacuum tube amplification to actuate a receiver; carbon instruments can actuate a magnetic receiver without vacuum tube amplification. "Midgets" have a low power capacity, but many aids have too much power for their receivers if turned on full. If a patient hears better at the telephone, he probably has a lesion of the middle ear and should be fitted with a magnetic air conduction receiver. Both the family and the personal history are of great help in making a diagnosis of the kind of impaired hearing. A contraindication to fitting an aid is otitis media purulenta chronica recurrens. A dry ear is *sine qua non*. Tests need not be made in a soundproof room when one is preparing to fit an aid. The patient is going to live in rooms with a greater or less noise level, and the examiner wants to know what the hearing is under such conditions. Halsted and Grossman classify hearing impairment into four types and use five means of determining the patient's functional loss. Six typical audiograms illustrate the method of procedure. In conclusion, they state: "It is better to inform the patient in advance of the benefits and limitations of a hearing aid in his special case, so that in the actual wearing thereof disappointment may be avoided."

VOORHEES, New York.

Nose

MANAGEMENT OF FRACTURES INVOLVING THE PARANASAL SINUSES. J. J. SHEA, J. A. M. A. **120**:745 (Nov. 7) 1942.

The author discusses fractures of the paranasal sinuses, with particular emphasis on adherence to the important principles and procedures followed in radical sinus surgery. Symmetric effects, a satisfactory bite, accurate replacement of bony fragments and adequate drainage of the injured sinus or sinuses are the immediate and final results to be obtained.

It is of the utmost importance that every patient should be hospitalized and observed for twenty-four hours and a consultation arranged with the neurosurgeon, if possible. The skin of the face should be cleansed immediately, and every particle of foreign material must be meticulously removed, hemorrhage controlled and the open wounds powdered with sulfanilamide.

Transfusion of blood or intravenous administration of an isotonic solution of sodium chloride is necessary to prevent shock in the more seriously injured. Not until the patient's general condition warrants the ordeal of an operation should corrective steps be undertaken.

From this point, the author describes some of his own technics in dealing with fracture of the maxillary sinus in relation to fracture of the malar bone, listing his approaches and the reasons therefor; he also discusses the management of fractures of the frontal, the ethmoid and the sphenoid sinus.

The paper was discussed by several rhinologists.

Dr. Robert H. Ivy, Philadelphia: Early reduction is urged whenever it can be done with safety, because if these fractures are allowed to unite firmly in malposition, satisfactory correction later may be impossible. One frequently sees cases of grave permanent facial deformity, with loss of function of the jaws, either because the condition of the patient did not permit early reduction or because in the anxiety over the cranial injury the facial deformity was entirely overlooked. For the diagnosis of fractures of the upper jaw and the malar bone greater reliance may be placed on clinical signs and symptoms. Roentgen examination of these regions is disappointing, and planograms have not given me much satisfaction.

In the case of the ordinary fracture of the malar bone with rupture into the maxillary sinus I do not think it is necessary or advisable to open and drain the sinus.

Dr. M. F. Arbuckle, St. Louis: It may be of interest to hear what my associates and I are doing in repair of these fractures in an army camp. A sergeant had lost about half his scalp and the rest of it was cut up as though some one had run a knife around his head. He had a fracture through the maxilla, with a downward displacement, so that his mouth could not be shut because the teeth would not line up. He had a fracture of the skull, loss of an eye and fractures of a leg, an arm and several ribs. He was unconscious and bled a great deal. He was put to bed, and nothing was done except to administer supportive measures. After several days the fragments of the jaw slipped back into position and remained there. I have been struck with the readiness with which fragments of the jaw, nose or skull will remain in position if they are replaced without much prosthesis.

The first principle of treatment is to put the soldier to bed and do as little as possible for the first week or ten days, aside from supportive treatment. The neurologist and the internist see him, but no extensive corrective measures are instituted until his life is no longer in danger. The most important single thing in the way of treatment is the use of a sulfonamide compound. We invariably give the drug to the limit of saturation.

GORDON, Philadelphia.

ACUTE FRONTAL SINUSITIS: THE TREPHINE OPERATION FOR DRAINAGE IN SELECTED CASES. LAWRENCE R. BOIES, Laryngoscope **52**:458 (June) 1942.

Boies recommends the use of a small trephine opening through the floor of the frontal sinus in cases of acute infection of the cavity in which drainage seems inadequate under a conservative regimen, as indicated by unrelieved headache or severe and progressive edema of the adjacent upper eyelid. He reports the successful use of this method of treatment with 4 patients. He considers it important that a relatively short time should elapse between the onset of the edema of the eyelid and surgical drainage. The interval in the cases reported was two, four, five and four days. The opening through the floor of the frontal sinus avoids cancellous or diploic bone, a precaution which reduces the danger of inciting osteomyelitis. The opening should be made posterior to a line extending upward from the posterior lacrimal crest, so that a small rubber tube may be inserted as a drain.

WOOD, Newark, N. J.

Miscellaneous

STREPTOCOCCOSIS IN CHILDREN. P. L. BOISVERT, D. C. DARROW, G. F. POWERS and J. D. TRASK, Am. J. Dis. Child. **64**:516 (Sept.) 1942.

The records of 4,849 patients from 1 to 16 years of age were studied at the New Haven Hospital. The children treated before the advent of sulfanilamide and its derivatives were classified as group I and the children treated during the three years since the introduction of this therapy were classified as group II. In nearly all cases the streptococcus in some form was found in the nose and throat, in suppurating lesions of the ears or glands or in the blood.

The favorable influence of sulfanilamide was shown in the low incidence of operative mastoiditis and meningitis, although many children had suppurative otitis media. Streptococcic fever of the childhood type suggests a "first" infection; scarlet fever is perhaps a manifestation of an allergic reaction to the organism. Follicular tonsillitis is typical of reinfection with a streptococcus and may be considered streptococcic fever of the adult type.

The authors recognize an "active" and a "latent" form of streptococcosis and speak of "sick" and "healthy" carriers, but admit that the connotation is not clear. Bacteremia is characteristic of a large majority of children who die of streptococcosis. In group I (in which no sulfonamide drug was used) the mortality was 9.6 per cent; in group II, in which sulfanilamide was given, it was 1.4 per cent, with no death in one of the years in which this group was studied.

The article contains graphs and twelve tables, in which the source material is analyzed. The paper is a highly important contribution to otorhinolaryngology, for it brings to light evidence that many of the so-called general diseases, such as scarlet fever, arise in the nose and throat and should be treated from that standpoint.

VOORHEES, New York.

THE EFFECT OF PROPYLENE GLYCOL VAPOR ON THE INCIDENCE OF RESPIRATORY INFECTIONS IN A CONVALESCENT HOME FOR CHILDREN. T. N. HARRIS and J. STOKES JR., Am. J. M. Sc. **204**:430 (Sept.) 1942.

Preliminary work has shown that the vapor of propylene glycol even in as high dilution as 1:50,000,000 is particularly effective for sterilization of air in enclosed spaces, the gas destroying with great rapidity air-borne bacteria and the human influenza virus A, as demonstrated by plating and animal tests. A great reduction in the incidence of infections of the respiratory tract was noted in a convalescent ward during a period of vaporization of propylene glycol in the ward. The concentration of the vapor in the air of the ward was shown to be in the effective bactericidal range. An inexpensive method for maintaining such a concentration in a ward or other enclosed space is described. These preliminary observations appear to confirm the effectiveness of propylene glycol vapor as an agent for sterilizing air in enclosed spaces. At a time when large numbers of men are assembled in military barracks, the additional importance of this method is obvious.

DAVISON, Danville, Pa.

OTOLARYNGOLOGICAL PROBLEMS OF AVIATION IN WORLD WAR II. PAUL A. CAMPBELL, Ann. Otol., Rhin. & Laryng. **51**:293 (June) 1942.

The problems involved are (1) proper selection of personnel, (2) effect of flight on hearing, (3) aero-otitis-media, (4) air sickness, (5) aerosinusitis, (6) effect of altitude on the voice and (7) effect of anoxia on the hearing mechanism.

1. From the point of view of the otolaryngologist, a candidate can become a good airman if he simply possesses an average nose of normal function, average ears and hearing and eustachian tubes and sinuses without obstruction. His balance mechanism must be within normal range and be without danger of vertiginous episodes. He can be trained to ventilate his ears on rapid descent; he can learn what illusions to expect from his balance mechanism and to rely on instruments for spatial orientation, and he can be told not to fly when he has nasopharyngitis or sinusitis.

2. Keeping the hearing at a high degree of efficiency is important. Changes in altitude and fatigue of the organ of Corti produced by the 80 to 110 decibels or more of noise present in flying result in a constant alteration in the hearing curve. This change takes place minute by minute. Changes in altitude resulting in a change in barometric pressure of 100 mm. of mercury can produce a fluctuation of several decibels of hearing in the conversational range, even between the acts of swallowing. Aircraft noise can produce a 15 decibel loss in the 3,000 to 5,000 frequency area in one hour's time and, in the experience of the author, a 25 decibel loss after four hours of flight. Most changes in hearing are transitory, and hearing usually returns to normal within a few hours after complete ventilation of the middle ear and quiet. Sufficient traumatism of this kind may, however, produce permanent changes.

3. Aero-otitis-media is a great problem. Ascent produces little difficulty, as the air in the mastoid cells and middle ear seeps through the eustachian tube easily between the acts of swallowing. On descent things are different, as most persons are able to equalize the then relative negative pressure of the middle ear only by swallowing, yawning or thrusting the jaw forward, as in a stifled yawn. The mechanism of this flutter valve effect is not entirely understood. It is believed that the relative differential pressure forces the epithelium of the lumen of the eustachian tube tightly together. If failure to swallow, or the presence of some anatomic condition or swelling of the epithelial lining, prevents the entrance of air, a negative pressure of from 100 to 200 mm. of mercury is developed and the epithelial lining is locked tightly together. As the pharyngeal muscles act only on the orifice of the tube, swallowing then is of no avail, and the end result is aero-otitis-media. When the negative pressure reaches 100 to 120 mm. the pain rather suddenly becomes severe and 10 or more decibels of hearing is lost in some portion of the low tone range (128 to 2048 double vibrations). The drum membrane is markedly retracted, and the vessels along the malleus are injected. Soon ecchymosis may appear in Shrapnell's membrane. Within from a half to eight or ten hours, serosanguineous fluid and bubbles form in the middle ear. After a while the pressure will be equalized by this fluid and the flutter valve released. Complete recovery may take from three days to three weeks. Fortunately, only a few ears become infected. Politzerization and catheterization are of little value unless performed immediately, and then are not always efficient. The most efficient treatment is to place the patient in a pressure chamber and return him to the level of pressure at which the block occurred, and then slowly return him to ground pressure, instructing him to swallow frequently. If the condition has existed over a period of hours, the only other alternative is the use of heat to increase the production

of fluid and analgesics to relieve pain and the application of shrinking medicaments to the eustachian orifice. The author feels that paracentesis is absolutely contraindicated. He calls attention, also, to the danger of an infection of the external canal spreading between the layers of the drum membrane, owing to the extensive and frequent excursions of the drum head which accompany the changes in barometric pressure and in the ventilation of the middle ear. This may produce first bullous myringitis and then a diffuse layer of pus between the layers of the drum membrane. He has seen no perforations follow.

4. Air sickness is caused by rapid changes in direction caused by changes in air currents, maneuvering, etc. No single simple remedy is known to relieve this condition completely.

5. Aerosinusitis has increased in frequency as greater altitudes and speeds of ascent have been reached. The mechanism is similar to that of the production of aero-otitis-media. Polypi or redundant tissue obstructs sinuses, and pressure in the cavities cannot be equalized. If the relative negative pressure within a sinus becomes great enough, the mucosa may tear away from the walls or submucous hematoma may result. Those with chronic sinusitis should not be selected for air crew duty; those with acute sinusitis or rhinitis should not fly.

6. At high altitudes, such as 35,000 feet (40.6 kilometers), the barometric pressure is 178 mm. of mercury, and the air passing between the vocal cords is less than one-fourth as dense as that at sea level, making speech difficult.

7. Anoxia affects the higher centers, in some respects in a way similar to that of acute alcoholism, and the interpretation of sounds is difficult. Work has been done in this field, and is being continued, but no definite report is yet available.

M. V. Miller, Philadelphia.

Tonsillectomy and Poliomyelitis. Editorial, J. A. M. A. **118**:980 (March 21) 1942.

The editor reviews the reports and comments of a number of investigators who observed the relationship of tonsillectomy to the development of poliomyelitis. The tonsillopharyngeal region seemed to show greater susceptibility, one of the investigators concluding that the virus progressed along local peripheral nerves to reach the nuclei of the cranial nerves.

The bulbar form of poliomyelitis was far and away more common in tonsillectomized persons and further suggested that the nasal pharyngeal mucosa is particularly vulnerable and may contain some additional factor which determines the outcome of exposure to the virus.

The conclusions arrived at are that since the time for tonsillectomy is practically always selective, it would appear desirable to have the operation performed during the season which does not coincide with the prevalence of poliomyelitis. This might eliminate a number of cases of the grave and highly fatal bulbar form of the disease.

Gordon, Philadelphia.

Caustic Effect of Sodium Sulfathiazole Solution on Nasal Mucous Membranes. C. E. Futch, L. K. Rosenvold and C. E. Stewart Jr., J. A. M. A. **119**:7 (May 2) 1942.

The authors conducted a series of experiments by applying solutions of sodium sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) in 5 and 30 per cent concentrations to the nasal mucous membranes of rabbits. Determinations of the hydrogen ion concentrations revealed that the p_H of both the solutions was approximately 10.

The studies indicated that these solutions had an injurious effect on the mucosa, causing destruction of the cilia and the superficial layers of the columnar cells, with failure of recovery even after one week.

The authors believe that the solutions were too alkaline, which is in full agreement with the recent observations of Fabricant, and they express the hope that nonirritant chemical combinations of the sulfonamide compounds may be made available and may prove useful in the future.

Gordon, Philadelphia.

Otolaryngologic Problems Occurring in Fliers. Leon D. Carson, J. A. M. A. **120**:4 (Sept. 5) 1942.

Carson discusses the effects of military flying and the punishing physical and mental strains that pilots are compelled to endure, emphasizing particularly the importance of the maintenance of optimum physical fitness.

He discusses the subject under five subheads: (1) the nasal structures and accessory sinuses, (2) the middle ear and eustachian tube, (3) the mouth and pharynx, (4) the acoustic function and (5) the vestibular mechanism.

Comments are made on adequate ventilation, nasal obstructions, allergic manifestations and, especially, the formation of polypi. Means of obtaining the best airway are also discussed. When surgical correction can be performed it should be done in order to permit free aeration. Polypi and chronic sinus disease are causes for rejection.

The question of "aero-otitis-media," as described by Armstrong, is fully entered into. It has been repeatedly observed that in fairly rapid descents from high altitudes the first part of the dive causes no discomfort, whereas acute distress will begin at between 15,000 and 20,000 feet (4.5 to 6 kilometers), or at lower altitudes.

There is little noticeable effect on the mouth and pharynx of fliers except those who have been habitual mouth breathers, and in such it has been found that more or less excessive dryness of the mouth and irritation of the pharynx are present.

The impairment of hearing caused by changes of pressure is a purely transient phenomenon. When hearing is impaired for a longer period as a result of prolonged flights, it is believed that the loss is due to fatigue of the acoustic function. Though this explanation is used to cloak the lack of exact knowledge of what produces the fade-out of hearing, it is hoped that further researches by competent physiologists, psychologists and engineers will soon reveal the ultimate causes.

The author concludes his discussion with a moderately detailed description of the functions concerned with the various phases of equilibrium and the relationship of the body adaptations to the movements encountered in flying.

He believes that the visual function is the most important, since it certainly dominates all the other functions. He also describes endolymph flow and illusions and refers to the question of vertigo and the development of the paradoxic use of the visual sense alone, to the exclusion of all other senses, whenever one must "fly blind." GORDON, Philadelphia.

TREATMENT OF RHEUMATIC FEVER. HOMER SWIFT and OTHERS, New York State J. Med. **42**:895 (May 1) 1942.

Many ear, nose and throat specialists have long been convinced that rheumatic fever and rheumatic heart are merely the general expression of a primary "streptococcic sore throat." Many internists are unconvinced. One of the latest books on the heart makes scant mention of any such connection. Dr. Swift states that the role of hemolytic streptococci in rheumatic fever is an important one. In Denmark both streptococcic infections of the throat and rheumatic fever are reportable diseases, and studies published by Danish workers seem to show a definite relation of rheumatic fever to epidemics of streptococcic angina. Green, of the British navy, found the incidence of streptococcic sore throat to be closely related to that of rheumatic fever. The high incidence of antistreptolysin O in the serum of patients with rheumatic fever seems to show that nearly all are suffering from hemolytic streptococcus infections. Swift states: "No single specific strain of streptococcus has been demonstrated as the cause of the disease [rheumatic fever], but many different types appear to be implicated." Moreover, no serums combat the acute forms; in fact, many antistreptococcus serums seem to have a pejorative effect. There is probably as much of the natural antistreptococcic antibodies in the system as nature requires during an effort to establish immunity. Removal of tonsils and adenoids does little good, for the infection has passed to all surrounding tissues and is in the cervical lymph nodes. That is why the sulfonamide drugs are not effective save in the early stages of sore throat.

It is indeed encouraging to have an internist of Dr. Swift's caliber state that "one emerges with a firm conviction that the etiology of rheumatic fever is closely linked to hemolytic streptococcic infections." VOORHEES, New York.

THERAPEUTIC PROCEDURES IN BRONCHIAL ASTHMA. W. C. SPAIN, New York State J. Med. **42**:1631 (Sept. 1) 1942.

Spain is interested chiefly in the allergic and medical aspects of asthma and describes five stages, the "sequence of phenomena," leading up to an open attack. Preventive treatment is feasible only if it is known that one or both parents are allergic; then a common cause may be established and the child protected accordingly. "Tonsils and adenoids proved to be infected should be removed, since they may harbor the organisms to which a bacterial sensitization may later occur." Spain then describes the acute, the chronic and the intractable forms of asthma. A procedure of importance is evacuation of infected material from the paranasal

sinuses, particularly the antrums, in acute attacks of infective asthma. He says: "Prompt relief may be noted within one or two hours."

Certain drugs are useless; others should be used with caution. The useless substances are calcium, histaminase and potassium chloride. The drugs to be used with caution are phenobarbital, acetylsalicylic acid, acetophenetidin, the salicylates, belladonna, with its alkaloid atropine, and morphine. Morphine may cause death from depression of the respiratory center.

A change of climate may become essential to avoid dust, smoke and fumes. This is especially true of children too young to undergo operation on the nose. Arizona and New Mexico are preferred because they have an average humidity of 35 per cent in summer and 65 per cent in winter, with a rainfall annually of only about 18 inches (20 cm.). Treatment in general is immunologic and specific.

VOORHEES, New York.

RESPIRATORY DERANGEMENT DURING ANESTHESIA. C. L. BURSTEIN, New York State J. Med. **42**:1638 (Sept. 1) 1942.

Burstein describes in detail the various "planes of surgical anesthesia" and illustrates the respiratory movements by means of graphs taken during various "states." He records a classification of derangements commonly observed during inhalation anesthesia and stresses the importance of the pneumograph in studying these deviations from normal breathing. The causes and management of apnea, hyperpnea, hypopnea, tachypnea, bradypnea, Cheyne-Stokes respiration, bronchoconstriction and obstruction of the upper respiratory tract are dealt with in detail. For the average clinician and surgeon the subject is somewhat technical, but it goes without saying that here is a subject that none can afford to neglect, since it is true, morally and legally, that the surgeon, and not any assistant at an operation, is entirely responsible for the life and well-being of his patient.

VOORHEES, New York.

"FIELD-FIRE" AND INVASIVE BASAL CELL CARCINOMA—BASAL-SQUAMOUS TYPE. JAMES B. BROWN and FRANK MCDOWELL, Surg., Gynec. & Obst. **74**:1128 (June) 1942.

The authors have observed a type of basal cell carcinoma that spreads through the derma without causing much surface ulceration and without producing much new tissue. It seems to be almost entirely destructive and to leave behind a healed area, but to present an ever increasing lesion with a small raised, advancing edge possessing intact or temporarily ulcerated epidermis. Microscopic sections from the edge and from any remaining areas of activity show basal cell carcinoma. To identify this growth clinically, the authors have called it "field-fire basal cell carcinoma." An important clinical point is that the growth may not appear serious; its diagnosis may even be missed, but in reality it is exceedingly malignant in that it may carry on by invasion primarily. When invasion has once become firmly established at the side of the nose, about the orbit or deep in the region of the ear, the growth is practically incurable. This point is important in prognosis and in teaching, as there has been a somewhat general idea that basal cell growths present little difficulty in treatment and cure. Moreover, the incidence of basal-squamous cell carcinoma of pure type may run as high as 20 per cent. Of all carcinomas, the basal cell type gives the best chance of cure by early removal, either by excision or by irradiation, but it is more dangerous to tissue and to life than is generally thought.

H. A. MILLER, Philadelphia.

Society Transactions

MASSACHUSETTS EYE AND EAR INFIRMARY

Selected Cases Reported at the Regular Weekly Clinical Meetings During 1942

ROBERT L. GOODALE, M.D., *Editor*

Mucocele of Frontal Sinus: Report of Three Cases. DR. H. G. TOBEY, DR. PHILIP MYSEL and DR. E. J. HALTON.

CASE 1

DR. E. J. HALTON: A white woman aged 68 entered the Massachusetts Eye and Ear Infirmary on Jan. 12, 1942 with a history of pain and swelling of the right eye of one year's duration. She had had "sinus trouble" for eleven years. In 1936 two polyps were removed from the right nasal cavity. One year prior to admission, after a "cold," there developed swelling of the eyelids and intermittent pain over the right side of the head. In the past few months this pain and swelling have been more severe.

The right eye presented swelling and ptosis of the upper lid, with narrowing of the palpebral fissure. Beneath the supraorbital rim there was a moderately firm, slightly tender mass which pushed the globe downward, outward and slightly forward. The upward and convergent motions of the right eye were limited. Nasal examination revealed nothing abnormal.

DR. A. S. MACMILLAN: Roentgenograms showed a depression of the rim of the right orbit and within the right frontal sinus a chronic suppurative process, surrounded by sclerotic bone.

DR. E. J. HALTON: The patient was operated on this morning by Dr. Mysel.

DR. PHILIP MYSEL: The woman's general condition was not good, and I elected to do the operation with local anesthesia. As Dr. MacMillan pointed out, I encountered a large mucocele, or rather a pyocele. It was filled with pus. The ethmoid cells were necrotic; the whole floor of the frontal sinus was paper thin, and I made a large opening from the frontal sinus into the nose and removed the middle turbinate and the ethmoid cells, but did not open the sphenoid sinus. There was a pulsating area about the size of a 5 cent piece in the posterior wall where the dura was exposed. The mucous membrane of the posterior wall of the frontal sinus attached to the dura was not removed in this case. I felt that if a large opening was obtained into the nose the danger of recurrence would be small.

CASE 2

DR. E. J. HALTON: A white man aged 53 entered the hospital five days ago (Jan. 16, 1942) complaining of pain and swelling of the right eyelids and the right side of the forehead. This had recurred three times during the past year.

About ten to twelve years ago a bilateral frontal operation was done at another hospital for relief of asthma. Except for occasional double vision, the patient had been in good health until about one year ago. Then he had a "cold" followed by pain and swelling of the right eye. This subsided. A few months later it recurred.

One week before admission he had severe pain and edema of the right eye and the right side of the forehead, which persisted until the day of admission. Examination showed equal pupils, an old scar of the left frontal area and ptosis of the right upper lid. In the medial inferior angle of the right orbit there was a soft, mildly tender, fluctuant swelling, pushing the eyeball downward and outward, with no notable exophthalmos. The motion of the eyeball was limited. The nose showed a defect of the right anterior ethmoid but no pus. Laboratory studies gave essentially normal results. Roentgenograms were taken on admission by Dr. MacMillan.

DR. A. S. MACMILLAN: The patient was operated on ten or twelve years ago. On the right side was a line representing a regrowth of cells in the old operative area in the frontal bone. The same process was taking place on the undisturbed side.

DR. E. J. HALTON: Because of the acute stage of the infection, warm boric acid dressings were employed for the external swelling and sprays of cocaine for the nose. The patient has improved remarkably.

CASE 3

DR. E. J. HALTON: A woman aged 63 was presented five weeks ago and again last week (January 14). She entered the hospital on Dec. 13, 1941. Physical examination showed exophthalmos of the right eye, with downward and outward displacement. Both lids were thickened.

DR. A. S. MACMILLAN: The roentgenogram showed an area of destruction involving the roof of the orbit, the sphenoid sinus and part of the lower and the left wing of the sphenoid bone. A cell entirely separated from the frontal sinus, and looking somewhat like a supra-ethmoid extension, was noted, but it had no connection with the nose or with the ethmoid labyrinth as far as one could tell. It involved the roof of the orbit and extended back as far as the apex of the orbit. Dr. Philip Mysel operated on the patient on December 19.

DR. PHILIP MYSEL: The patient presented destruction in the roof of the orbit and invasion of the sphenoid bone. She had all the clinical signs of an orbital abscess. The question was whether a tumor was present in the orbit and whether the infection was superimposed on this tumor. Dr. MacMillan's diagnosis was a mucocele. I exposed the orbit from above through an eyebrow incision, not carrying the incision down onto the nose as far as usual with the radical frontal operation. A good-sized abscess was evacuated. Culture of material from the abscess showed Staphylococcus aureus.

I found that the dura was exposed. A large collection of granulations filled the lateral wall of the nose and the orbit as far back as the orbital fissure. The os planum appeared normal. I packed the cavity loosely with some gauze impregnated with sulfanilamide powder. Dr. Tobey and I discussed whether, after the acute phase had subsided, we were justified in opening this wound and making an attempt to drain this area of destruction through the nose. We concluded it would be impossible to do this without taking the eye out; so we have left the situation as it is. We are allowing the cavity to fill with granulations.

SUMMARY OF THREE CASES

DR. H. G. TOBEY: In the first case repeated infections of the frontal sinus had eventually shut off the nasopharyngeal duct, and a mucocele had developed. In the second case, in which the condition was postoperative, a mucocele had probably begun to form, and the third case was one of a probable mucocele in the posterior wall of the ethmoid. The cases also illustrate the various means of treating mucoceles in this region. Perhaps the ideal treatment is complete removal of all the mucous membrane in the lining of the cyst. This is not always possible, of course, particularly when the lesion is attached to the dura, although it is still possible to touch up the mucous membrane with phenol or with coagulation diathermy. But if there is good drainage through the nose, the growth will probably never recur, as Dr. Mysel stated. The last case, of course, illustrates another way of taking care of a mucocele, that is, by leaving the wound open and letting it granulate from the bottom. This can be done in a small, shallow frontal sinus, but not in a large one, especially one that has been enlarged by the spreading of the mucocele.

DR. G. L. TOBEY: I cannot understand why the mucocele in the third case, if it is in a posterior ethmoid cell, could not be drained through the nose.

DR. H. G. TOBEY: If you had seen it, I do not think you would have considered opening the right ethmoid cell. It was completely blocked off from the rest of the ethmoid system. The ethmoid plate was intact. There was no question in my mind that the proper procedure was to let the cavity granulate. The capsule of the eye has helped to obliterate the entire area.

Suppurative Acute Otitis Media of the Right Ear, with Mastoiditis and Petrositis.

DR. H. G. TOBEY, DR. PHILIP MYSEL, DR. F. W. OGDEN and DR. R. L. GOODALE.

CASE 1.—A white boy aged 6 years was admitted on March 11 with a history of a discharging right ear for three weeks, following an infection of the upper respiratory tract. Sulfadiazine (2-[paraaminobenzenesulfonamido]-pyridine), given by his local physician about a week after the drainage started, decreased the amount and relieved the patient of pain during the time he was taking the drug.

Five days before admission to the hospital pain again developed in the right ear and also appeared in the temporal region. One day before admission the discharge from the right ear suddenly increased with decrease of the pain. His temperature was 101 F., and the white blood cell count was 10,900, with 67 polymorphonuclear cells and 24 lymphocytes.

Local examination showed that the right drum membrane was red and thick and bulged slightly. There was a postcentral perforation. A large amount of thick yellow pus was present. Postaurally there were definite tenderness and some questionable thickening over the

region of the mastoid antrum. The tonsils were moderately enlarged. Neurologic signs were negative. Roentgenograms were taken by Dr. MacMillan.

DR. A. S. MACMILLAN: The right mastoid was rather thoroughly involved. It was large, with a pneumatic petrous bone, but the petrous apex itself showed considerable decalcification. The petrous ridge seemed almost defective at one point. The roentgenogram taken through the orbit showed the apex completely decalcified. A roentgenogram taken at the base showed rather thorough excavation of the right base. There was a swelling in the nasopharynx, suggesting an infection of the petrous apex as well.

DR. F. W. OGDEN: On March 12 a simple mastoidectomy was performed on the right side.

DR. PHILIP MYSEL: Operation revealed that the mastoid was large, badly infected and well broken down. I was careful to exenterate the mastoid cells completely. The sinus plate was intact and solid. I did not uncover that. I did uncover the dura of the middle fossa to make sure there was no extradural abscess. I opened the antrum sufficiently to see the short process of the incus, and then proceeded to exenterate the solid angle to see whether there was a lead into the petrous pyramid. I worked out the posterior canal and the superior canal and the cancellous bone between these canals. I found no direct lead into the petrous pyramid. I exposed the inner cortex of the inner plate of the mastoid, which was white and intact.

In view of the absence of meningitis, I felt I could temporize for twenty-four to forty-eight hours to see what would happen. I filled the cavity with sulfanilamide powder, packed it loosely with iodoform gauze and returned the patient to the ward. He did very well. He vomited now and then, but I felt that was probably due to the medication. I stopped the administration of the drug by mouth after three or four days, and he stopped vomiting. He had no abnormal neurologic signs, and the temperature was normal. The headache was gone. I think that when we take another roentgenogram of the petrous bone in three weeks there will be a different picture. In my opinion, the process is of a congestive type, and I think the area will then have a more normal appearance.

CASE 2.—This 7 year old girl has been presented before. She was admitted to this hospital on Feb. 26, 1942, with a history of bilateral simple mastoidectomy two and a half years ago. She had been well until one month before the present admission, when a postaural abscess developed on the right side during an infection of the upper respiratory tract. Her local physician examined her, and two weeks before admission she came to the outpatient department with a postaural discharge.

DR. A. S. MACMILLAN: The roentgenogram showed that cells still remained on the affected side, and there was definite involvement of the right petrous pyramid. The outline of the roof of the right petrous pyramid was gone at one point, whereas the roof on the left side was intact.

DR. F. W. OGDEN: The patient left the outpatient department and returned to her local physician, who revised the simple mastoidectomy cavity on the right side and placed the patient under chemotherapy. The recovery from this revision was apparently uneventful, and the patient went home three days before her admission to the Massachusetts Eye and Ear Infirmary.

Two days before admission to the infirmary photophobia and headache developed, with some diplopia; one day before admission she had nausea and vomiting. She was seen in the clinic just before her admission to the house, when there was first degree nystagmus to the left. This was not seen when she was admitted to the ward. The temperature on admission was 102 F. There were squinting and closing of the right eye at the time of admission, and she complained of double vision. The diplopia was most apparent when she looked to the left side. The right ear drum was thick, and there was a thick discharge in the canal. No landmarks were seen on the drum membrane. Postaurally, just opposite the region of the antrum, there was a fistula, with a fetid discharge. The patient had a moderately stiff neck at this time, with a questionable Kernig sign. Cultures of material from the ear showed Staph. aureus.

On lumbar puncture the initial pressure was 225 mm., with 125 leukocytes in the spinal fluid, 85 of which were polymorphonuclears. The white cell count was 15,500. Administration of sulfadiazine, which had been stopped after she had returned home from the other hospital, was resumed. On February 27 the simple mastoidectomy cavity was revised by Dr. Mysel.

DR. PHILIP MYSEL: I examined this patient on the morning after admission. She had had diplopia on the previous afternoon, but on the day I saw her she had no paralysis of the sixth nerve and little stiffness of the neck. However, examination of the spinal fluid showed beginning septic or irritative meningitis.

I opened the mastoid and found that the previous mastoidectomy had been incompletely done. The sinus had been exposed at that operation. I removed the sinus plate, opened the drum wide and worked out the solid angle, as in the previous case. It was a question in my mind whether to do a radical mastoidectomy or not. If I decided to do a radical mastoidectomy I should look for a lead anteriorly, and if I did not find such a lead I should remove the base of the petrous pyramid and enter the petrous pyramid by way of the solid angle. I chose not to carry out either of these procedures because the patient did not look sick. I did not feel like sacrificing the middle ear at this time. I decided to give the patient twenty-four to forty-eight hours and see what would happen; I filled the cavity with sulfanilamide powder and packed the cavity loosely with iodoform gauze. The cells cleared up; the meningitis cleared, and she made an uneventful recovery. I think another roentgenogram was taken for comparison with the first.

DR. A. S. MACMILLAN: The break through at the roof of the petrous pyramid seemed to be about the same as that observed previously.

DR. PHILIP MYSEL: I don't think one can base one's surgical approach on the roentgenographic evidence alone. One must have some clinical basis for a decision to enter the petrous pyramid. This patient still has an open postaural wound. I shall leave it open for one week, and if she is all right I shall then make a plastic closure. I feel that a roentgenogram taken two weeks from now will reveal a different picture. The middle ear is now dry.

DR. P. E. MELTZER: Here in one morning 2 cases of petrositis have been reported, and up to ten or twelve years ago I don't think any one here would have recognized such a condition. It would have been said that the cause of the otitis was an incomplete mastoidectomy alone.

Dr. Mysel stated that he did not have a reason for doing a radical operation. Certainly, chemotherapy has given the surgeon more courage. With the evidence that he had, with two operations and meningitis, I should elect to do the radical mastoidectomy. I still am concerned about the petrositis.

DR. H. G. TOBEY: There is still the middle ear on which we can depend for symptoms. If the middle ear does not clear up, the reason for it is clear.

DR. PHILIP MYSEL: The second roentgenogram was better than the first, and I have a feeling the third one will be even better.

FURTHER REPORT, MAY 6, 1942

DR. E. J. HALTON: On March 27, a month later, a secondary plastic closure was done, and the patient was discharged on April 9. She was perfectly well; the postoperative wound was dry, and the middle ear and the drum head were entirely normal until April 30. She came back to the hospital the next day complaining of headache and transient dizziness, which passed off by the middle of the morning. Last Friday the dizziness became severe, with nausea and vomiting.

On her admission to the hospital, physical examination showed a retracted drum membrane, with the postaural wound intact and no swelling or evidence of infection; there was first, second and third degree nystagmus to the right.

DR. A. S. MACMILLAN: The roentgenographic examinations taken while she was still in the House did not show much change, and in the last one the cavity seemed to be filling in. Comparison of the last roentgenogram with the one taken at the time of the present admission showed that the process had advanced. The area of the petrositis is again much decalcified.

DR. E. J. HALTON: The child had no hearing in the right ear on admission. On lumbar puncture there were 37 polymorphonuclears, a clear fluid and normal dynamic readings. Sulfadiazine therapy was started. Last Saturday night the mastoidectomy cavity was revised.

DR. R. L. GOODALE: This patient had labyrinthitis in 1939, when she was first operated on. From that time she was apparently without symptoms, until reinfection took place, and she was readmitted on Feb. 26, 1942 for a revision of the mastoidectomy cavity. The dura at some time had been extensively exposed, practically from the posterior superior angle forward into the zygomatic process. The lateral sinus had not been exposed. The solid angle had been entered and considerable curettement done.

At the last operation, five days ago, the postaural wound was puffy and red. When the wound was opened a large cavity filled with pus around the solid angle and the mastoid antrum and a great deal of softened bone between the solid angle and the mastoid tip were observed. This was all cleaned out at operation. The solid angle was still further exenterated, so that the cerebellar cortex was completely exposed, and the procedure carried right down

toward the posterior semicircular canal, which was visualized and skeletonized. Soft bone was encountered most of the way in that direction. Then the lead appeared to stop. I decided to do a radical mastoidectomy. The incus was not to be seen. The malleus was taken out and the drum membrane removed. Granulations were present in the middle ear, and there was a lead superiorly, just above the facial nerve as it enters the middle ear. This lead was followed inward about ⅛ inch (3 mm.). There it ended. The facial nerve was exposed in this region by the progress of the disease, and in use of the very smallest curet, fairly severe facial twitches were observed. I think this patient has trouble farther in, but no other leads were observed at the time.

In spite of roentgenographic evidence of disease, the child has not had symptoms pointing to involvement of the petrous apex. The patient has no symptoms now, no facial paralysis, no headache. There has been no facial pain or disturbance of the ocular muscles, and I felt that the intervention was sufficient at that time. She has a wide open wound now, through which further operation can be carried out if necessary. I think that the keeping of her wound open for a longer period and repeated roentgenographic examinations constitute the proper procedure now. A lumbar puncture has not been done since the operation. I took the pack out today. The cavity is absolutely dry, with no pus whatever. The nystagmus has entirely disappeared.

DR. PHILIP MYSEL: This patient came in when I was on service. When I saw her she had mild meningeal irritation. She had been operated on at another hospital, and she had been put on sulfadiazine therapy the day before. On the day I operated the meningeal symptoms had cleared up. She did not look sick. The roentgenogram of the petrous pyramid taken at this time has been described. She had no clinical signs of petrositis, and I revised the mastoidectomy cavity at this time, cleaned it out and thoroughly exposed the incus, which must have sloughed out at operation, with exposure of the canal. She did very well. When I saw her off service, about nine weeks since the operation, the postaural wound was open. Now she is back with labyrinthitis and more decalcification in the petrous pyramid. Evidently, the ear has not been doing well since 1939, and I wonder whether we shall not have to intervene further.

DR. L. A. SCHALL: What about her labyrinth?

DR. PHILIP MYSEL: I think the labyrinth is dead. I should be inclined to do a labyrinthectomy, for two reasons: First, she has had meningeal irritation associated with definite labyrinthitis and second, the petrous process is destroyed anterior to the labyrinth; by doing the labyrinthectomy one would obtain more operating room.

FURTHER REPORT, MAY 13, 1942

DR. E. J. HALTON: After Dr. Goodale did the radical mastoidectomy on the right side, on May 2, the postoperative course was uneventful for four days. She was under sulfadiazine therapy, and last Wednesday, one week ago, we stopped treatment with the drug; on Thursday afternoon there developed weakness of the right side of face, with questionable swelling of the right zygomatic area. A lumbar puncture showed normal dynamics and 18 cells, 8 polymorphonuclears and 10 lymphocytes per cubic millimeter. On the following day, May 9, the right petrous process was explored by Dr. Goodale and Dr. Lurie.

DR. R. L. GOODALE: Last week the possibility of going in further and exenterating the decalcified area in the petrous apex was discussed. At that time I said that I felt we could safely keep the patient under observation for a few days and operate if we had to. Now, in a review of the history, a few factors appear which I did not know of at the last meeting.

When the patient was here in February she had had slight diplopia. There had been no facial pain or obvious involvement of the fifth nerve, but there was evidence of irritation of the sixth nerve. At the last admission she had an exacerbation of an infection or irritation of the labyrinth. There were nystagmus and vomiting. The temperature was elevated, and it seemed best at that time to do a radical mastoidectomy. The facial nerve was exposed, and there were a few twitches of the face when the nerve was touched.

There was no weakness of the facial muscles until five days after operation, when definite weakness of the right eyelid and the right corner of the mouth appeared. The aural discharge lessened a great deal. All this time we were watching for other signs of involvement of the petrous pyramid to appear clinically. She denied having any diplopia. Hearing returned after the last radical operation. I did not use the noise apparatus in testing, but she did hear in the right ear. I think she has a live labyrinth.

At the last operation a lead was observed anteriorly around the eustachian tube which led inward toward the apex. The facial nerve was observed. More granulations were cleaned out between the facial nerve as it enters the middle ear and the eustachian tube,

and a very large area of decompression was made near the carotid canal. The carotid artery was exposed, so that it could be retracted anteriorly, and a very small curet was passed down into the carotid bony canal and plunged into the petrous apex. A cavity was encountered in this region, which was full of soft, diseased bone, but there was no frank pus. I think that the absence of pus can be explained on the basis of the therapy with sulfadiazine she was receiving. The facial nerve twitched a great deal during this last operation.

She can close the eye still, and she can smile with that side of the mouth, although when she shows her teeth the strong pull of the left side overpowers the muscles of the right. She has had no increase in temperature, no nausea and no vomiting. Clinically she has done well since the operation. The cavity was filled with sulfadiazine powder, and a drain inserted down to this region, but not into it. The drainage is difficult, as the carotid artery is in the way.

DR. M. H. LURIE: I wish to add that the medial plate of the carotid canal was not soft. In other words, Dr. Goodale and I had to go through hard diseased bony plates before we reached the petrous tip. Now the question remains whether the disease will clear up, or whether there is some infection around the labyrinth which may necessitate a labyrinthectomy. At present we shall sit tight and watch.

DR. PHILIP MYSEL: This little girl came under the care of my service during the winter. She had had two simple mastoidectomies, one in 1939, which was incomplete, and one a few weeks before her admission, and still the mastoidectomy had been incompletely done. She was admitted with mild meningeal symptoms and the roentgenographic changes described. The postaural wound was draining, and I revised the mastoid cavity at this time. There was a question of my doing a radical operation at the time of the revision. I decided to wait two or three days and see if she would get along without such an operation, for I did not want to sacrifice the middle ear. She had a little diplopia, but no true paralysis of the sixth nerve or pain in or around the eye. She was not sick. I did an extensive simple mastoidectomy and instituted treatment with sulfadiazine for three days; then its use was discontinued. From that time convalescence was uneventful. She was placed in the ward for weeks. We made roentgenographic examinations every ten days, and kept the wound open.

On March 31 the petrous pyramid showed definite improvement. She had no labyrinthine symptoms during the period of observation. Then the postaural wound was closed. She was observed for a week or ten days and then discharged to go home.

She returned with acute destruction of the labyrinth, in my opinion. The house officer said the hearing was good at the time. This patient had a third degree nystagmus.

The roentgenogram shows that the destruction is along the tegmental surface, and not along the surface between the transverse part of the carotid artery; with this destruction of the labyrinth, I feel better results would be obtained if exenteration were done. Such an exposure is advantageous if this disease is in the petrous pyramid or in the part anterior to the labyrinth, as the roentgenogram shows. I should think the chance of cleaning out the diseased bone would be better. That the facial paralysis came on four or five days later, and not immediately after the middle ear was cleaned out, also favors the idea that this paralysis is secondary to disease in the labyrinth. The process is a long-drawn-out one, and I may be wrong in not having done the radical mastoidectomy earlier. We had a similar case, that of a boy with a completely decalcified petrous pyramid, with a high initial pressure, in which I did a similar operation and carried out conservative treatment; this patient up to date has shown no evidence of any intracranial disease, and there is still decalcification.

The question is: Should we do a radical operation on this girl now, or should we have done it earlier? With destructive labyrinthitis with meningeal irritation, I should utilize the labyrinthine approach to the osteomyelitic area, in preference to the anterior wall. It is a great mistake to think that meningitis will not develop in the presence of a viable labyrinth. This patient may not be out of danger yet.

DR. L. A. SCHALL: The value of the meetings is to welcome the differences of opinion of different members of the staff. A further report on the progress in this case is awaited.

FURTHER OBSERVATIONS, DEC. 16, 1942

DR. R. L. GOODALE: After her discharge from the hospital on July 3, the patient made an uneventful recovery. Roentgenograms taken on July 29 showed that the area of destruction in the petrous had filled in with healthy bone. There has been gradual diminution of the discharge from the ear, and at present (December 16) the operative cavity is dry.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLARYNGOLOGY, AND PHILADELPHIA LARYNGOLOGICAL SOCIETY

GABRIEL TUCKER, M.D., *Chairman*

LOUIS J. BURNS, M.D., *Chairman*

WILLIAM HEWSON, M.D., *Clerk*

THOMAS F. FURLONG JR., M.D., *Secretary*

Regular Meeting, Feb. 17, 1943

GEORGE L. WHELAN, M.D., *Editor of Abstracts*

The Problem of Impaired Hearing. DR. JAMES A. BABBITT.

It is obvious that a paper with a title of such far reaching scope should attempt to bring up to date and to analyze the present situation with respect to impaired hearing. The subject might be discussed under three heads: (1) the discovery and management of hearing disability in children of preschool and school age; (2) the enormous number of mature, but still active, persons who are handicapped by deafness in their economic and social relations, and (3) the increasingly large number of deafened persons in the geriatric, or older age, group.

It will probably come as a pleasant relief to this audience to learn that there is no intention of reducing this paper to another monotonous and tiresome review of the procedures in the examination of thousands of school children, which statistical reports from state boards of health and education have already covered and which have been shown in country-wide surveys; neither is it intended to delve again too deeply into the ultrascientific investigations of otosclerosis, which have not yet arrived at a satisfactory differential diagnosis, or even to discuss the captivating subject of fenestration for quick relief in the impeded ear. In the effort to by-pass the conventional procedure, I have selected for this brief paper three or four topics which seem to have escaped in a measure the usual themes selected for the reams of addresses and publications on the subject of impaired hearing.

Certain generalized themes relating to the prevention of deafness have been presented, and most adequately, in recent years.

The prenatal influence of certain drugs, for example, quinine, the salicylates and alcohol, has been investigated by such clinicians as Covell, Taylor and Mosher, and from the standpoint of the ear the action of these drugs would seem of even more importance than simple maternal nutrition. It seems to have been definitely established that the placenta is no barrier to the drugs, which readily enter the fetal circulation, may be toxic to the fetus and cause nerve deafness. It may be a far cry from prenatal prevention to child, school and college health administration, but its importance should be recorded.

From available records it is safe to assume that 10 per cent of school children have defective hearing as yet undiscovered. In a certain proportion of children this deafness cannot be cured, but there are a gratifying percentage in which the handicap may be removed and a larger percentage in which it can be prevented. But note this carefully: Except for a comparatively few persons who are born deaf, and a few with a family predisposition to deafness, this defective hearing, formally ascertained by school examination, could have been discovered and its progress prevented while the child was solely under home care, from birth to early school age, and this prevention could have been aided by the pediatrician and the alert family physician.

It would seem to be the duty of the otologist, in his part of the war effort, to step out of his office chair and enter the campaign to bring about, perhaps through his efforts in large measure, a gratuitous service to his country. It is his function to spread the gospel of recognition of early symptoms of deafness in every contact, in every time and in every place possible, to place parents on the alert to observe simple signs, such as oral and obstructed nasal breathing, mental dulness, inattention to conversation, slow reactions and aloofness in the play hour, turning of the head to listen and traumatism to the ear in rough play. Like the adult, the child is sensitive to his own defect. Perhaps the parent or the physician should study the family tree. The physician should watch with double care the convalescence from and complications of such diseases as scarlet fever, measles, mumps, diphtheria, influenza and pneumonia. One should never make light of persistent colds or joint and muscle pains, whether or not one accepts the danger of focal infection anywhere in the body. Probably the common cold is the most difficult to manage, with its problems of ventilation and, as is

now believed, of detrimental chilling of night air. It should be remembered that, aside from bacterial contiguity, one is most likely to take cold in a draft when one is fatigued or overheated. The warning of a running nose should not be neglected. Finally, the otologist should convey to parents that their country demands 100 per cent normal eyes and ears.

In studying a series of patients with tinnitus, the manufacturers supplied me with prostigmine, as they have other investigators in this city, in order that I might make a considerable test of its value in treatment. Every desirable method of care was combined with its use, in an endeavor to get an impression of it. In this sense, of course, the experiment was not an isolated test of the product.

Injections of greater or less frequency were given to 53 patients, about 450 injections being administered in all. The results were tabulated, but as this was not with thought of publication, and as so many injections were given in connection with other treatment, their report would have little research value. Of these 53 patients, 16 did not have over 3 injections, and the results will not be considered in the estimate of the value of this drug. Two of this number were benefited, but the good effects must have been from the associated help of the other methods.

Of the remaining 37 patients, 2 manifested remarkable improvement. These 2 patients had rather intensive treatment and carried it on for a considerable time under home physicians. Each had approximately 40 injections and took prostigmine bromide with varying regularity. The tabulated results follow:

Total number of patients in series	53
Patients discarded because of limited number of injections, 3 or less	16
Patients considered in study	37
Patients showing remarkable improvement	2
Patients with definite but moderate improvement	7
Patients whose improvement was uncertain	7
Patients showing no improvement	21
Patients with temporary deterioration	4

Of a total of 9 patients who showed improvement, the tinnitus was much relieved in 7, unchanged in 1 and worse, for some reason, in the last. I was anxious to get the history of tinnitus because of the optimistic report of a New York observer, but my results were not encouraging in this respect.

DISCUSSION

DR. GEORGE M. COATES: This fine paper of Dr. Babbitt's is difficult for me to discuss because he has taken up the problem of deafness from so many angles. I shall, therefore, remark only on some points which interested me most.

If Dr. Babbitt's patients had acute or subacute deafness, almost any treatment would have helped them, or they might have improved without any treatment at all. If the deafness was of long standing, his results are remarkable.

According to most statistics, the results of the use of prostigmine are not good. That corresponds with my experience. A few patients have been benefited, but many have not. Dr. Babbitt referred to the investigations of Houser, Campbell and Schleuderberg, who reported that there was little value in treatment with prostigmine.

I do not agree with Dr. Babbitt that tubal treatment is on the discard. I believe that in many cases of deafness tubal treatments cannot be given up. If deafness is due to an allergic condition, I treat the allergy. In many cases of deafness, even those of long standing, the patient can obtain relief from tubal treatment.

The examination of the ears given by the draft boards is inadequate. They pass many patients whose hearing is impaired and reject many whose hearing is good. They make mistakes, or perhaps cannot devote sufficient time to the examination.

I hope this paper by Dr. Babbitt will stimulate much discussion. Many fine papers go by without discussion. There are many points in Dr. Babbitt's paper that could be elaborated on with profit.

DR. HENRY PLEASANTS JR., West Chester, Pa.: It is presumptious for a mere physician to attempt to discuss such a paper as Dr. Babbitt's. I was impressed with its thoroughness. He has called many things to our attention that we as otologists should take home to our communities.

He referred to some of the work in the detection of hearing defects in the college of which I am the medical director. Some of the points he mentioned may be worth discussing.

My fellow examiners and I carry out complete audiometric tests on all the students in the college, the maximum number being 1,600. We have had reports from group audiometric tests

which were checked at the infirmary. We have not noted that a large percentage of students enter college with impaired hearing. I feel safe in saying that 90 per cent have good hearing. The improvement in the condition of the students who have impaired hearing as a result of expert care has been phenomenal.

In 1939 we rendered 13,300 health services at the infirmary. We endeavored to cut down the dust menace. The next year the number of health services rendered was 10,300; last year it was 7,300. Not all colds were due to dust, but elimination of the dust hazard has been effective in reducing the number of colds. The number of cases of disorders of the ear has materially diminished. Another important measure is a campaign among students in the prevention of scarlet fever and such contagious diseases.

Many teachers are susceptible to scarlet fever; they are not immune. You as members of this society represent the very top of the specialty of otolaryngology. Perhaps you can bring the weight of your education to bear on the boards of trustees of teaching institutions for young people in your communities so that they may know how important is the conservation of the hearing, vision and general health of the youth of the nation, and so that the incidence of deafness of more than 43 per cent will not occur again in 1961.

DR. WARREN B. DAVIS: The time limit will permit me to discuss only the treatment with prostigmine. I began this treatment with a number of patients when prostigmine was first popularized. My most outstanding cases of improvement were those encountered in the first few months of trial with the drug.

The most remarkable case was that of a music teacher who had been deaf for sixteen years. Her hearing was of much interest to her, since her livelihood depended on her hearing sounds. I was not able to understand why her hearing was so improved.

Sixteen years before she had had both drum heads ruptured by her husband, when he came home one night and slapped her on one ear and then on the other. Her hearing improved 25 or 30 per cent after treatment with prostigmine, and she also lost her tinnitus. The improvement was maintained. At present I do not see her more than two or three times a year.

DR. ROBERT F. RIDPATH: I am interested in the subject of prostigmine because an associate of mine was one of the men who first reported on the drug. We tried it on only a few patients with aural disorders, as my work is mostly concerned with the sinuses. The number of patients I have treated is small. Dr. Davis asked me to give my patients a series of treatments. I tried such a series on several persons and obtained no improvement, except in 1 patient. The result in the case Dr. Davis mentioned was phenomenal. Dr. Davis insists the injections should be given three times a week. We followed this through on a patient, who showed much improvement after a series of treatments, after a decided impairment in hearing for four years.

A person who smokes a large number of cigars or cigarets every day may acquire an allergic condition and a hearing defect which increases greatly with the use of tobacco. All know that after four or five cocktails the hearing is less acute. Alcohol has a deleterious effect on the hearing.

We obtained some improvement in several patients with impaired hearing by treating intestinal toxemia. Although we have had little experience with prostigmine, we have had several decided cases of improvement from injections and oral administration of another product in patients approaching the menopause. Administration of ovarian extract or testosterone propionate for two or three months will, I believe, give some good results.

DR. MATTHEW S. ERSNER: I congratulate Dr. Babbitt. My associates and I have used prostigmine for chronic deafness. We did not employ it with the acute or the subacute form because a great many patients with such deafness improve spontaneously. With 69 patients we failed. To the patients who thought they improved with treatment of the ear we gave physiologic solution of sodium chloride, and they still felt that their hearing was improved. Other patients were tested at different times of the day. In the morning their hearing was better than in the evening. At noon there was a drop in the acuity of hearing. We were not successful with prostigmine therapy.

DR. JAMES A. BABBITT: I appreciate Dr. Coates's allusion to acute deafness. None of my patients had acute deafness except for 1, who had unilateral deafness, but prostigmine did not have anything to do with its cure. Dr. Pleasant's mention of the dust menace was interesting. I thank you all for the generous discussion.

The Hearing Aid from the Patient's Point of View. DR. WALTER HUGHSON and MISS EVA THOMPSON, Abington, Pa.

This article will appear in a later issue of the ARCHIVES.

DISCUSSION

DR. OSCAR V. BATSON: Dr. Hughson and Miss Thompson have presented an interesting report on the work being done in the otologic research laboratory of the Abington Memorial Hospital. It might be called a report of progress, for I am sure that they would not have one feel that their studies are ended. I note in the several papers which have come from this laboratory a gradual change in the point of view as additional studies are made. It is difficult to discuss this paper, based as it is on exact observations. One can only ask for more facts and perhaps suggest alternative interpretations.

Without a consideration of medical statistics and the errors of sampling, the figures presented may be taken at their best face value. It is reported that 80 per cent of the fittings for hearing aids were satisfactory. This appears to mean that after careful analysis of the entire hearing problem, and the final selection of a hearing aid by the physician, 1 out of 5 patients did not agree with the results. This low proportion of dissatisfied patients is still too large to be looked on complacently. The difficulty might be with the otologist, with the hearing aids or with the patient. Possibly all three are involved. The authors try to examine the problem from the standpoint of the patient; I should like to mention some other points of view. First may be considered that of the otologist. It has been stated that to test the patients in the method devised and reported, apparatus costing no more than a good audiometer is required. There is much euphemy in this statement. To the beginner in otology this means that he must have twice the money necessary for the purchase of an audiometer before he can begin fitting hearing aids by this method. It means, further, that he must have a trained assistant to operate the microphone and control the level of the amplifier, so that a constant level of voice current is fed into the soundproof room. In addition, there is the cost of two soundproof rooms. Next, the otologist must acquire fourteen or more varieties of hearing aids. I should like to ask the total cost of these. If this total expenditure is to be made by the otologist, the cost must be added to that of his equipment and eventually passed on to the patient. It would be valuable to know the total number of man hours required to test a patient, that is, the total time and the number of persons required to examine a patient's state of hearing, to fit him with a hearing aid and to retest him for the efficiency of the aid after it has been delivered.

From the point of view of the manufacturer, there must be a minimum, which will vary from time to time, below which the cost of the hearing aid to the patient cannot fall. If approximately $20 is taken as the cost of unmatched parts used to make a hearing aid, one can double this to allow for the cost of assembly and other fair costs of production and double it again to arrive at a retail price which is commonly used in the surgical and scientific instrument trade. This probably represents a minimum price at which a hearing aid can be marketed today. There are hearing aids on the market, or were until recently, that were selling at about this price, or lower. It is true that some of these instruments were of the alarm clock variety, while the patient wanted a twenty-one jewel wrist watch, but so far as the acoustic service was concerned, several of them were adequate. The cost to the average patient seems too great, however; even the present cost, prorated over three or four years, with the upkeep included, is at the level of monthly telephone charges. A few years ago I was told by the head of a large hearing aid sales office that 50 per cent of the time of the office was consumed in helping the patients secrete the hearing aids about their persons. This increases the cost to the patient. If the cost of parts, as indicated here, is $19, it is obvious that the price of a hearing aid will never be at the level of the "clinic" eyeglasses. The cost, however, should eventually compete with the cost of an artificial set of full dentures. As to the advertising campaign being conducted at present by certain makers of hearing aids, I cannot be too pessimistic. For one thing, hearing aids have been increasing in number on the streets. This, I believe, is due to the improvement in hearing aids and to the fact that advertising has been directed toward increase in "consumer acceptance," as they say in the trade; this indirectly reduces the cost to the patient.

As to the hearing aids themselves, the studies of Fowler, of Rieger and of Pohlman and Krantz indicate that, even with nerve deafness, a hearing aid with uniform amplification should give adequate results. The difficulty has been that this uniform amplification brings loud sounds into the pain level. It is possible even now to limit the loudness level, but this adds to the size of the equipment. With the conductive type of deafness, straight line amplification seems to be highly satisfactory, so long as there is sufficient amplification. In my limited experience, patients having hearing aids equipped with a quality or tone control find this useful in adjusting to the size of the room in which they find themselves. All would appreciate it if the authors would point out which phases of fitting are most useful. Some of the manufacturers would have the otologists and the patients believe that it is necessary to

make the amplification curve a reciprocal of the curve of hearing loss, but, as noted before, this has not been the general opinion among otologists, and I should like to hear this point discussed.

Professor Pohlman pointed out to me a number of years ago that patients complained of tinnitus when they had a hearing loss for the speech range of about 20 decibels, and that they first became conscious of their deficiency in hearing when they had a loss of about 35 decibels for the speech range. With this loss I have found that a patient will not commonly accept a hearing aid. I should like to ask at what loss in decibels for the speech range the authors have found patients willing to accept a hearing aid.

DR. JULIUS WINSTON: I enjoyed Dr. Hughson's interesting paper on the use of an instrument that most people are reluctant to try. Many people with a 60 decibel loss do not want it. I believe that tinnitus is not to be expected to disappear with use of a hearing aid.

DR. WALTER HUGHSON: I am disappointed that Dr. Batson's chief concern is about the price of this aid. I have not had the time to consider the value or expense of the parts. It seems to me there is a tremendous waste all along the line. The general feeling is that the present price of hearing aids is exorbitant.

Dr. Batson brings up the question of how the average person is going to get equipment for these tests. I am fortunate because I have an experimental laboratory. It takes a great many hours to select the proper aid and to know that a proper job has been done for the patient. We do not believe that any hearing aid is good for all types of loss. In general, a characteristic aid is the best if a person can wear it.

We have stated that a 25 to 40 per cent gain in speech reception is the range we require for an adequate aid and that it corresponds to the need exhibited in a 40 to 60 decibel loss. A hearing aid can modify tinnitus only by its temporary masking effect.

Book Reviews

A Manual of Otology, Rhinology and Laryngology. By Howard Charles Ballenger, M.D., F.A.C.S., Associate Professor of Otolaryngology, Northwestern University School of Medicine, Chicago. Second Edition. Cloth, $4. Pp. 334, with 114 engravings and 3 color plates. Philadelphia: Lea & Febiger, 1943.

There has always been a need for "handbooks" and "outlines" in all specialties. This is especially true of otorhinolaryngology, the more "comprehensive" works on which run to 500 pages and more. In his new edition Dr. Ballenger has rearranged the material and amplified the text. The chapter on laryngeal conditions dependent on "nerves" required reediting, and it was necessary to add a chapter on foreign bodies in the lower airways. The technic of tracheotomy ought to be well rehearsed by every one from time to time, as the operation is a life-saving measure which calls for immediate action. Some four pages are devoted to it in this edition.

The "busy practitioner" has no time for theoretic discussions, and he cannot expect to learn surgical technic from a textbook. Only in an operating room can he apply the knowledge which he has gained from watching other surgeons at work. Dr. Ballenger knows the value of a thorough grounding in pathology and the use of illustrations. He gives just enough discussion of the pathologic changes involved.

The author is expert in condensing information. In about a page of printed matter he sets forth the basic principles to be remembered in use of the sulfonamide compounds. Student physicians, student nurses and the general practitioner will find much of value in this book. The budding otorhinolaryngologist may well use it as an introduction to the specialty he is seeking to master.

News and Comment

AMERICAN BOARD OF OTOLARYNGOLOGY

The next examination of the American Board of Otolaryngology will be held in Chicago, at the Palmer House and the Illinois Research Hospital, on Oct. 6, 7, 8 and 9, 1943. Candidates should apply at once to Dr. Dean M. Lierle, secretary of the board, University Hospital, Iowa City.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Claude C. Cody, 1304 Walker Ave., Houston, Texas.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.

American Academy of Ophthalmology and Otolaryngology

President: Dr. James A. Babbit, 1912 Spruce St., Philadelphia.
President-Elect: Dr. Lawrence T. Post, 508 N. Grand Blvd., St. Louis, Mo.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Place: Chicago. Time: Oct. 10-13, 1943.

American Broncho-Esophagological Association

President: Dr. Robert L. Moorhead, 125 Ramsen St., Brooklyn.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago.

American Laryngological Association

President: Dr. Charles J. Imperatori, 108 E. 38th St., New York.
Secretary: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.

SECTIONS:

Eastern.—Vice President: Dr. Wesley M. Hunt, 33 E. 68th St., New York City.
Southern.—Vice President: William C. Warren Jr., 478 Peachtree St. N. E., Atlanta, Ga.
Middle.—Vice President: Fred W. Dixon, 2060 E. 9th St., Cleveland.
Western.—Vice President: Dr. Simon Jesberg, 500 S. Lucas Ave., Los Angeles.

American Otological Society

President: Dr. Wesley C. Bowers, 17 E. 61st St., New York.
Secretary: Dr. Isidore Friesner, 101 E. 73d St., New York.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.